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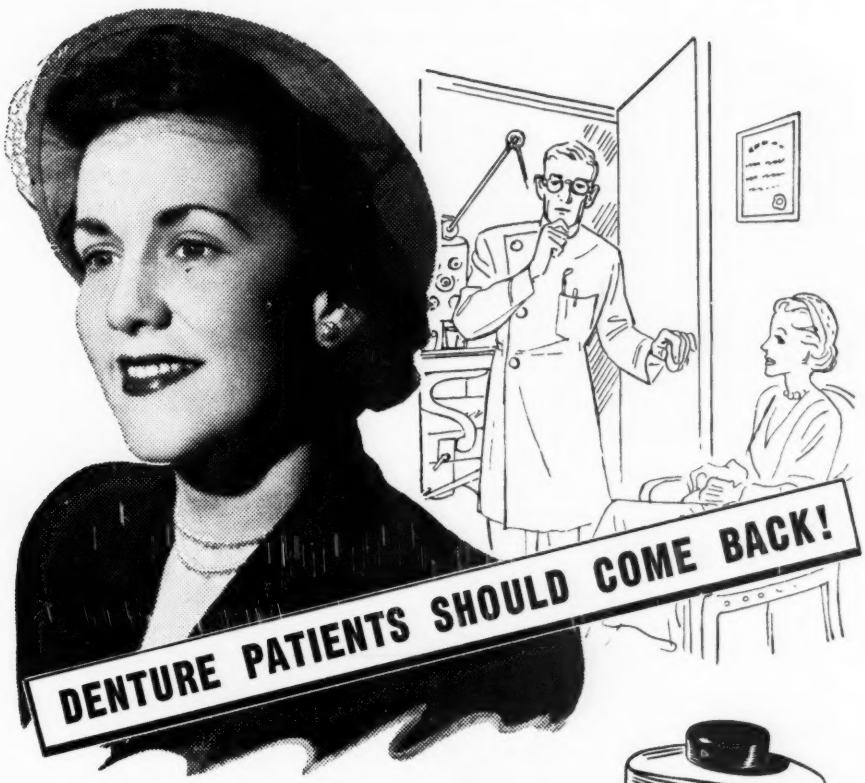

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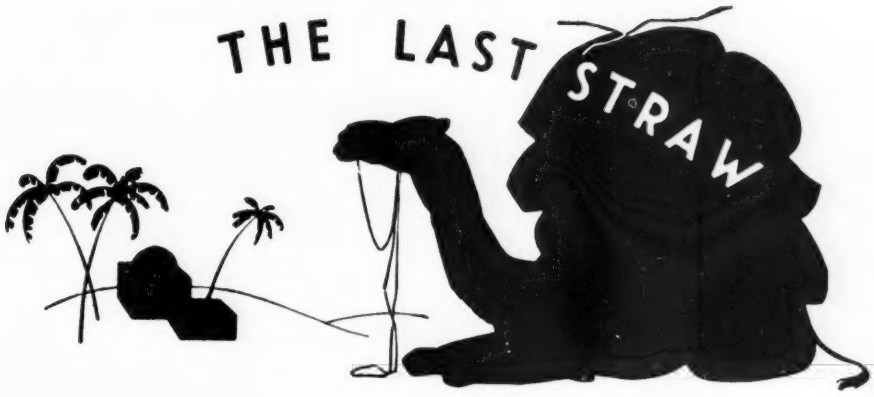

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The Composition and Structure of Human Dental Enamel*

H. R. Sullivan, D.D.Sc. (Syd.)†

Enamel is known to be a hard, rather brittle, calcified tissue. Its colour varies from yellowish-white to greyish-white depending primarily upon the degree of translucency of the enamel. It forms a protective covering for the dentine over the crowns of all normal teeth. Chemically, it has been consistently shown to contain (in dry weight) about 95% of inorganic ash and about 1.7% of organic matter. These figures, however, give a rather false idea of the possible importance of the organic matter because, by volume, the reticular portion of the organic substance (or stroma) is believed to occupy a space more nearly comparable with that occupied by the inorganic substance. The classical comparison of a stone and a sponge well illustrates this point[1]: a stone and a sponge may have the same volume but their weights vary greatly, the weight of the sponge being less than 1% of that of the stone.

The Structure of Normal Enamel.

Both the inorganic and organic materials found within human dental enamel form a structural pattern which in normal teeth varies only slightly, whereas quite marked differences are seen in the enamel of different animals.[2]

The main constituents of normal human enamel are the enamel rods or prisms, prism sheaths, interprismatic substance, enamel cuticle and tufts and lamellae. Within the enamel occur certain phenomena caused by the arrangement of the constituents, such as the striae of Retzius and the Hunter-Schreger lines.

Enamel prisms (Figs. 1-4).

Enamel prisms were first described by Retzius in 1837. It was soon realised that the enamel was fibrillated and could quite readily be split into long, calcified rods. These rods have been described as being built up of a row of calcified discs slightly separated from one another. Widdowson[3] termed these discs "varicosities" and stated that those of one prism lie side by side with those of its neighbours and do not interdigitate. Thus an appearance of cross striation is produced. Bodecker[4] described the prisms as columns of calcified substance which as a rule possess a hexagonal shape. Nasmyth[5] had thought that the structure of the prism was not so much a column as a series of cells arranged in regular rows and forming composite fibres placed nearly at right angles to the surface of the dentine. However, the concept of the columnar form is now universally accepted.

The pattern of a cross section of the rods varies in different parts of enamel and in different teeth. Round, square, hexagonal and heptagonal forms have all been described in addition to a peculiar "arcade" form.[4-7] Faber[8] suggested that the polygonal types are found in the outer enamel layers and Eisenberg[9] stated that they also occur in the incisal portion of the enamel. At times the existence of arcade-shaped forms has been denied. Meyer[10] suggested that it is an artefact, whilst Wolf[11] stated that it is a form brought about by displacement of the prisms. Gustafson[12] believed that the arcade forms are artefacts due to one side of the enamel sheath staining more readily than the other. Hopewell-Smith[13] put forward the suggestion that a perfectly formed enamel prism is hexagonal in transverse section because it is normally surrounded by six other prisms, whilst Gustafson[12] said that:

> Where the interprismatic substance is well developed the prisms are always round or nearly round. The hexagonal prisms only occur in such places where the interprismatic substance is poorly developed and the prisms consequently lie close to each other.

The majority of investigators[10, 14] believed that each prism passes from the dentino-enamel junction to the enamel surface as a discrete entity. Mummery[15] doubted this possibility and even Gottlieb[16], in 1947, suggested the presence of branching or supplementary rods, but it is the common belief that these do not occur. If, however, there are no supplementary rods, some explanation must be made to account for the variation in surface area at the dentino-enamel junction and the enamel surface. This has been explained by the fact that there is a variation in the width of the enamel rods at the various levels of enamel.

Measurements by Pickerill[17] indicated that the relationship of the diameter of prisms at the dentino-enamel junction to the diameter at the outer surface of the enamel is 1 to 1.83 and that measurements of the two surfaces involved indicate a relationship of 1 to 1.76. Thus, he suggested that there would be no room for the existence of secondary or supplementary prisms. He stated that the prisms increase in diameter immediately over the cusps more than elsewhere.

*Based on portion of a thesis submitted to the University of Sydney in support of his candidature for the degree of Doctor of Dental Science.

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Williams[18] stated that the ratio of the diameter of the prisms at the dentino-enamel junction to that at the surface is 5.5 to 10.

Despite these observations Jaccard and Grosjean[19] still maintained that there is little variation in the diameter of prisms and so they assumed that inserted supplementary prisms must fill out the interspace.

The average width of a prism is about five microns.[13] Scott and Wyckoff[20], using electron microscopy, confirmed the general appearance of the cross section of the rods and gave measurements of from 5μ - 10μ as the diameter of the prisms.

The enamel prisms manifest transverse striations which are seen quite regularly throughout the length of the rod. They can, however, be more easily observed in a decalcified or etched section and they lie some 3μ - 4μ apart.[21] They were originally described by Nasmyth[22] in 1841 who, however, thought that they were merely artefacts. The appearance of these structures has been interpreted as indicative of a rhythmical calcification. Firstly there is the phase during which inorganic material is mainly deposited and secondly there is a rest phase during which organic material is deposited.[23]

Andresen[24] has suggested that the cross striations bear some relationship to the interprismatic substance, whilst Bodecker[25] believed them to be offshoots from the prism sheaths. As will be seen later, Bodecker at this time used the terms "prism sheath" and "interprism substance" to denote the same substance, so that he is probably in agreement with Andresen. Their appearance has been claimed to be a sign of incomplete calcification or decalcification by external factors.[26] Forshufvud[27] has interpreted these striations as a massing of organic substance to form a diaphragm in the enamel prism. The striations can be seen quite clearly under polarised light.[28]

The interior of the prisms was originally considered to be homogeneous but some fibrillar structures have been described within them. Gustafson[12] described the presence of spiral threads passing through the prisms at an angulation of approximately some 50 degrees. His observations were based on the examination of polished and etched sections of enamel examined by reflected light. Other writers, mainly of earlier periods, have reported similar structures.[29]

They are believed to be of an organic nature. Quite recently Frank[30], reporting the results of an examination of enamel structure by means of the electron microscope, spoke of a fine network of ultra microscopic fibrils within the prism but connected to the prism sheath. Using electron microscopy, Scott, Ussing, Sognnaes and Wyckoff[31] described a submicroscopic fibrillar network which was almost universally present within and between the enamel prisms. They did not see any spiral organic structures within the prisms.

Frisbie, Nuckolls and Saunders[32] have propounded the idea that each enamel-forming cell is responsible for the formation of a single organic rod and its adjacent inter-rod substance which subsequently undergo a progressive mineralisation. They claim to have shown that this underlying protein matrix remains within the enamel at least up to the time of the eruption of the tooth. If this assumption be correct, it could have an important bearing upon the interpretation of the microscopic appearance of early carious lesions.

Prism sheaths (Fig. 5).

The description and investigation of prism sheaths was carried out principally by Bodecker[1] who demonstrated a sheath rich in organic material surrounding each enamel prism.

Rosebury[33] and Bodecker and Gies[34] produced evidence that the sheaths are made up of a keratinous substance. Their permeability to dye-substances and the possibility of lymphatic flow through them have already been discussed.

Chase[35, 36] using Bodecker's celloidin decalcification technique, supported the contention that the material is of the nature of a keratin and also stated that the organic content is present as often, and in as great amounts, in old enamel (up to 48 years) as in very young enamel (3 months to 1 year). This does not support the earlier investigations of Hoppe-Seyler[37] who considered that the enamel of infants sometimes contained as much as 14% of organic matter compared with the normal 3.6% in the enamel of adults. Gustafson[12] said that the two halves of the sheath are unequally developed, one half being more easily stained and more acid-resisting than the other half. This has already been mentioned as a possible explanation of the arcade forms.

Their existence has recently been confirmed by the work of Scott, Ussing, Sognnaes and Wyckoff[31], using electron microscopy.

Interprismatic substance (Fig. 6).

Baumgartner[38], along with others, suggested that enamel prisms lay in close apposition with each other without any interposed substance but this idea has long been discarded. Although the presence of a cementing substance is now accepted, some diversity of opinion exists concerning its nature. It was described

Fig. 1.—A transverse section of enamel in which the enamel prisms or rods can be seen extending throughout the length of the field. The striated effect, giving the appearance of "varicosities", is quite marked. It is not common, however, for such striations to be so obvious in normal enamel. (X 120. Transmitted light.)

Fig. 3.—A longitudinal section of enamel showing marked cross striation of the enamel prisms. In this photomicrograph the striations of the various prisms are lying adjacent to those of the adjoining prisms. (X 120. Transmitted light.)

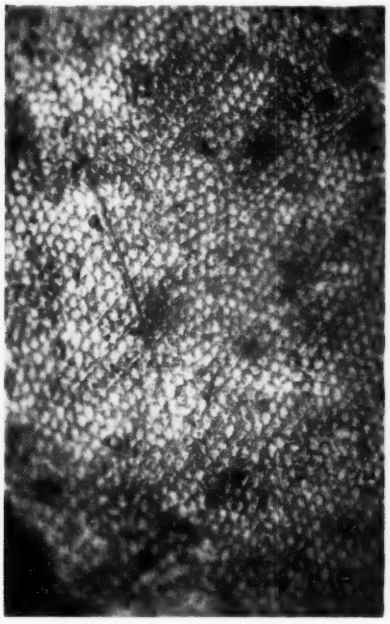

Fig. 2.—An enamel surface stained lightly with silver nitrate. It will be seen that the general outline of the prisms is roughly hexagonal but numerous deviate forms exist even in this field. (X 340. Incident light.)

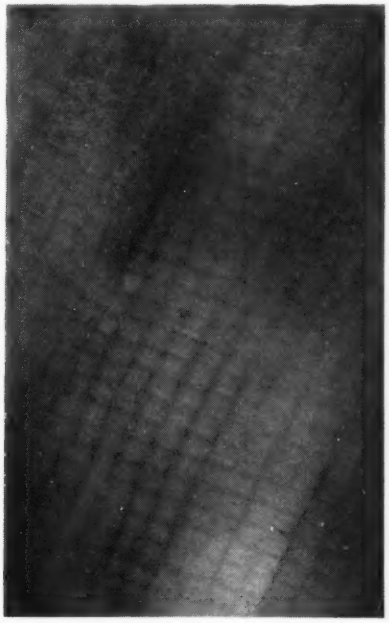

Fig. 4.—A higher magnification of part of the section shown in figure 3. (X 504.)

by Malleson[23] and Fish[39] as a material which could not be stained, and Jaccard and Grosjean[19] believed it to be homogeneously calcified. Other investigators, however, amongst whom was Bodecker[40], believed that this substance is easily stained. However, it should be noted in this regard that Bodecker speaks of "the interprismatic substance or enamel prism sheaths." It can thus be seen that the reason for some of the diversity of thought upon this matter is probably the fact that a number of the earlier investigators failed to make any distinction between the prism sheath and the interprismatic substance. Applebaum[41] also speaks of the "interprismatic substance, or organic matrix" being stained. This confusion probably arose, firstly, because interprismatic substance is often quite meagre; secondly, because it is difficult to demonstrate its presence except in sections which are cut through the prisms at right angles.

The distribution of the interprismatic substance throughout the enamel seems to vary greatly. Andresen[42] thought that it is better developed at the dentino-enamel junction. The variation in its distribution is mentioned by Gustafson[12] who stated:

> Where the interprismatic substance is well developed the prisms are always round or nearly round. The hexagonal prisms occur only in such cases where the interprismatic substance is poorly developed and the prisms consequently lie close to each other.

Bodecker[43] has postulated that one can normally see interprismatic substance far better in the teeth of young subjects and that it more or less disappears in older enamel. It is difficult, however, to understand how one of the portions of the enamel could be lost without any change in the form of the enamel.

It has also been pointed out that the interprismatic substance has a greater refractive index than the substance of the enamel rods and that it is more soluble in acids. Because of these facts it is possible to distinguish the individual structure of rods when they are viewed in unetched, longitudinal or transverse sections. Likewise, when a thin section of the enamel is cut parallel with the direction of the enamel rods and then subjected to the action of a solution of 2% hydrochloric acid, it will be seen that the interprismatic substance is attacked more readily; the acid dissolves it out from between the enamel rods and attacks their sides. Eventually the ends of the rods will be seen to project as they become freed from the surrounding interprismatic substance.[26] Noyes and others also noted that the acid produced in a carious lesion dissolves the interprismatic substance to a greater extent than has been possible by laboratory methods.

Chase[44] claimed that the interprismatic substance plays no part in the increase of the surface area of the enamel at the free surface as compared with that at the dentino-enamel junction.

Gustafson[12] failed to observe double refraction in the interprismatic substance and declared that the property of double refraction which other workers have ascribed to the interprismatic substance is actually a property of the prism sheath, which, he states, shows strong double refraction.

Scott and Wyckoff[20] examined replicas of enamel surfaces by electron microscopy and showed that rod ends are separated from each other by from 1μ — 3μ of interprismatic substance. The thickness varies quite considerably in a single area and on different sides of a single rod end.

Enamel tufts and lamellae.

It has long been the custom to group tufts and lamellae together, primarily because of the fact that they both have a relatively high organic content. Enamel tufts occur at the dentino-enamel junction and therefore presumably cannot play a part in the earliest stages of carious lesions. On the other hand, lamellae may pass through the entire width of the enamel.

Enamel tufts (Figs. 7 and 8). Enamel tufts arise at the dentino-enamel junction and may penetrate the enamel for up to one-third of its thickness. Originally they were thought to resemble tufts of grass in form but Orban[45] and Beust[46] have shown that they are ribbon-like structures arising from the dentine. The plane of the ribbon lies in the long axis of the crown. They are composed of hypocalcified enamel rods and interprismatic substance.

The original idea that tufts might be artefacts produced by cracking of the sections during preparation was quickly refuted when the use of decalcification techniques for the study of the organic structure of the enamel became common.

Forshufvud[27] has described them as consisting of a comparatively thick fibril running into the enamel for a short distance and not branching out into more than one or two ramifications. They are seen only in transverse sections and their numbers vary according to the type of tooth and the position within the enamel.

Those who have considered enamel to possess some circulatory system have often identified the tufts as one of the main tissues through which such a system might function[46], particularly as quite early their appar-

ent connection with the dentine was demonstrated in teeth decalcified *in toto*.[47]

Gustafson[12] maintained that the tufts are sites of deficient mineralisation which are much more extensive than is indicated by staining. He said that these "areas" divide the enamel into alternately narrow, fairly soft layers (the tufts) and broad, harder layers (between the tufts). They consist of less mineralised but, for the rest, normal enamel substance and are consequently not a new structure in the architecture of the enamel. He suggested that such an arrangement would be advantageous, both from the mechanical and nutritional point of view.

Enamel lamellae. As the enamel tufts, so too the lamellae were considered by many of the earlier workers to be merely cracks or artefacts introduced into the enamel during the preparation of a specimen. However, it can be quite readily shown by decalcifying a ground section and watching the process under a microscope that they are organic structures extending from the dentine to the enamel surface.[48]

It has been suggested that lamellae are malformations and consequently cannot be counted among the normal structures of the enamel[49], but Beust[50] affirmed that their regular appearance in apparently normal enamel warrants their inclusion among normal anatomical structures.

Some workers[17, 51] have linked tufts and lamellae together on the ground that the lamellae are merely tufts which extended to the outer surface of the enamel. However, Orban[52] denied this relationship.

Fish[53] considered that lamellae are formed by the inability of some ameloblasts to lay down calcium salts and so, instead of prisms, residues of organic substances are found.

Frisbie and others[32] believed that lamellae are groups of prisms in which both the maturation and the calcification of the matrix are incomplete.

The most widely accepted concept of lamella-formation[48] is that planes of tension develop in the maturing enamel. In some cases the tissues in these planes do not calcify and in others actual cracks occur which are then filled in from the soft tissues surrounding the crown. Thus, two types of lamellae are formed: those composed of poorly calcified prisms and interprismatic substance and those composed of different tissue elements which have invaded the cracks from without during the pre-eruptive stage. The first of these types is always limited to the enamel, whereas the second may extend even into the dentine. According to the form of the lamellae, it is possible to obtain a hornified, outer, secondary cuticle in the cleft[54], or to have a deposition of cement.[48]

Gustafson[12] still maintained that the lamellae are almost identical with tufts. He suggests that the dentinal portions are misinterpretations of cracks which allow dye to penetrate into the dentine. He stated:

> The lamellae must be considered as tufts which have retained a tendency to less mineralisation during the whole development of the enamel.

Enamel spindles (Fig. 9).

In ground sections examined by transmitted light, a series of fairly thick projections can be seen extending from the dentino-enamel junction for a short distance into the enamel. These occur primarily around the cusps of teeth and, because of their shape, they have been termed spindles.

There is little variation in expressions of opinion concerning the formation of these spindles and it is generally accepted that they originate from processes of odontoblasts and extend into the enamel before the enamel is calcified. They usually lie at right angles to the surface of the dentine and therefore are not always parallel with the enamel prisms.[48] These formations are of little importance as they do not arise from ameloblastic tissue.

The enamel cuticle.

A delicate membrane normally covers the crowns of newly erupted teeth. Because it was originally described by Nasmyth[5] in 1839, it was called Nasmyth's membrane. It is only approximately 1μ in thickness[55], and it has been suggested that it is continuous with the lamellae.[53] Earlier workers considered that this membrane consisted of only one layer which was formed by the ameloblasts after their production of the enamel rods had been completed. This view has now been modified and it is believed that there are two layers known as the primary and secondary enamel cuticles. The primary cuticle is believed to be formed from the ameloblasts, whilst the secondary cuticle arises from the reduced enamel epithelium which covers the crown. The primary enamel cuticle usually calcifies but it may be more resistant to acids and alkalis than the true enamel.[48] The secondary enamel cuticle, however, is of a keratinous nature, which has been amply demonstrated by its marked birefringence in polarised light.[28, 56]

It is generally assumed that mastication and other mechanical influences lead to the loss of the cuticle from most portions of the crown

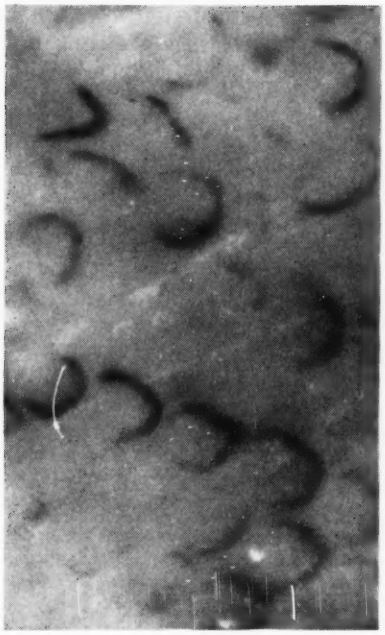

Fig. 5.—A tangential section of enamel stained with silver nitrate. The prism sheaths are seen as dark areas surrounding the unstained prisms. (X 1,000. Incident light.)

Fig. 7.—Enamel tufts arising at the dentino-enamel junction. (X 504. Transmitted light.)

Fig. 6.—A tangential section of enamel stained with silver nitrate. Spaces can be seen between the stained prism sheaths which, presumably, contain interprismatic substance. (X 1,000. Incident light.)

Fig. 8.—A transverse section of enamel showing numerous tufts arising from the dentino-enamel junction. (X 175. Polarised light.)

soon after the eruption of the tooth. It is possible, however, that in protected areas, such as the approximal surfaces or fissures, the cuticles may remain intact throughout life. In this regard it might be noted that Pincus[57] described a "groove" protein in connection with his theory of the etiology of dental caries. He has also identified the secondary cuticle as a scleroprotein.

Gustafson[12] had great difficulty in isolating the two cuticles—indeed so much so that he stated:

> Although I did not succeed in observing any secondary membrane, I have the impression that the authors which (*sic*) have described it earlier are in the right.

Gottlieb[16] drew attention to work previously reported by Bodecker which indicated that a connection exists between the cuticle and the enamel lamellae and the prism sheaths.

Widdowson[3] made an attempt to analyse the structure, origin and function of Nasmyth's membrane. His conclusions upon the structure and origin are in keeping with those already reported and his remark upon the function is that it is unknown. He referred to various works which indicate that the role of the membrane may be to act as: (i) a protection against acids; (ii) a dialysing membrane, or (iii) a medium in the formation of the epithelial attachment.

The striae of Retzius (Fig. 10).

In 1837 Retzius[6] described the appearance of brownish, parallel bands in ground sections of the enamel. They can be seen best by transmitted light. In longitudinal sections they can be seen passing from the cervical margin in oblique fashion from the dentino-enamel junction to the surface of the enamel, deviating occlusally as they approach the enamel surface. In a transverse section of the enamel the striations appear as concentric circles. From the first they were considered to be poorly calcified zones within the enamel and also to be an indication within the fully developed enamel of "pauses" that may have occurred during the development of the enamel. Schour and Hoffman[58] suggested that the cross striations of the enamel prisms appear more clearly in the striae of Retzius and that the intervals between the striae are in multiples of sixteen, from 32μ—128μ.

Any idea that the striae were formed by a layer of pigment was disproved by the work of Applebaum and his colleagues[59] and of Hollander and others[60] who, using Grenz rays, showed that the striae illustrate the successive apposition of layers of enamel matrix during the formation of the crown. They concluded also from their results that there is a difference in density of calcification between the striae and the remainder of the enamel.

Barker[61] also believed that the striae indicate the border line between different layers of calcification.

Orban[48] described the striae thus:

> They may be compared to the growth rings in the cross section of a tree. The term "incremental lines" designated these structures appropriately, for they do, in fact, show the advance of growth of the enamel matrix. The incremental lines are an expression of the rhythmically recurrent variation in the formation of the enamel matrix.

He pointed out that metabolic disturbances can cause a pathological thickening of the striae rendering them more prominent.

Gustafson[12] claimed that he could not demonstrate any variation in hardness between the striae and the surrounding enamel. He also stated that investigations using polarised light indicated that the rhythm of cross striation of the prisms does not seem to be disturbed in the striae. He suggested that the striae are caused by a displacement of the prisms during the development of the enamel which is manifested by "bends" in the prisms facing towards the cemento-enamel junction. This view, however, is not in accord with that of the majority of workers.

Sognnaes[62] used decalcified paraffin sections to examine the zones in which striae of Retzius were observed. He reported that:

> The organic framework has a pattern of its own in the regions of the incremental lines, which . . . satisfactorily explains the morphological appearance of the incremental lines of the enamel.

He showed that the prism sheaths are actually thickened at the sites of the striae. Remembering Bodecker's confusion between prism sheaths and interprism substance, this latter observation is probably in agreement with the earlier observations of Bodecker[1] who indicated that the inter-rod substance is thickened at the site of the striae at the expense of the prisms.

The Hunter-Schreger lines (Figs. 11 and 12).

These optical phenomena were observed and described over one hundred years ago. They appear in longitudinal sections of the enamel as broad bands passing from the vicinity of the dentino-enamel junction in a slightly curved line towards the enamel surface. The convex surface is always towards the gingival margin of the tooth. They can best be seen when viewed by reflected light and appear as alternating dark and light strips of varying width. Orban[48] declared that in a sagittal section the prisms are generally cut obliquely and if the bundles of rods are traced from the surface of such a section into the depth it will be observed that they run obliquely in one disc

to the right, in the next disc to the left. This concept was supported by the work of Gustafson.[12] The results of investigations in which soft x-rays[60] and vital staining[63, 64] were used indicated the possibility of some variation in the calcification of the enamel which is coincident with the distribution of the Hunter-Schreger lines. This was also supported by the observation of Orban[48] who stated that by careful decalcification these alternate zones could be shown to possess slightly increased permeability and a higher content of organic material. However, Gustafson[12] could not show any difference in the mineralisation of the different zones.

Staz[65] suggested that these bands or lines indicate some stages in calcification. Erausquin[66] stated that they correspond to a hexagonal double curvature of the prisms in the inner two-thirds of the enamel in the lateral plates and involve a complete ring of enamel. In different rings, the deviation is to the left or right alternately. Sognnaes[67], using a low temperature demineralisation technique, has shown that when viewed in a longitudinal plane the densely packed prism sheaths which are arranged in longitudinal, corrugated segments give rise to the pattern which makes the Schreger bands.

Composition of Enamel.

The inorganic constituents.

Because of the nature of the enamel, it is comparatively easy to enumerate quite accurately the majority of its contents. This can be done because enamel is so highly calcified that the investigation of its composition amounts primarily to an analysis of inorganic salts. Some variation occurred in the results of earlier investigations but more recent observations[68-74] have shown remarkable consistency. However, all investigators reported that some variation occurs in the concentrations in individual specimens and thus it is difficult to do other than accept a statistical figure for what might be called normal enamel. It has also been suggested that the concentration of the salts varies, according to the site on the tooth. The following may be accepted as average figures: calcium 35.5%, phosphorus 17.5%, magnesium 0.4%, and a calcium/phosphorus ratio of 2.1.

Inorganic salts other than those already mentioned are present in smaller amounts but on that account should not be ignored, because the presence of trace elements in biological material is often of fundamental importance. Sodium, potassium, lead, zinc, iron, copper, chlorine, fluorine, lithium and strontium have all been shown to be present quite consistently in normal enamel.[69, 75-80]

Whilst it has been a comparatively simple matter to decide upon the basic constituents of the inorganic portion of the enamel, it has been by no means a simple task to determine the form in which the inorganic enamel substance exists. Bassett[81] originally showed the ability of calcium and phosphorus to form a stable, basic phosphate—3 $Ca_3(PO_4)_2$. $Ca(OH)_2$—with a crystalline structure characteristic of the mineral apatite. The apatite structure, because of its stability, was accepted as the type most likely to exist in bone and by analogy it was assumed that it is also the form in which the inorganic salts exist in enamel. This observation was supported by the fact that the calcium/phosphorus ratio for $Ca_3(PO_4)_2$ is 1.94, whilst that for hydroxyapatite is 2.15, a figure which is very similar to that obtained for enamel; also by the work of Taylor and Sheard[82], who showed that the refractive index of enamel is 1.62 and that of apatite minerals is 1.63.

The difficulties associated with the study of such a highly calcified tissue are great, but the technique of x-ray diffraction proved to be most valuable and gave apparently accurate results. X-ray diffraction patterns for enamel and mineral apatites were observed and compared by Cape and Kitchin[28], and Thewlis.[83, 84] Bale and others who all agreed that great similarity exists between the structure of enamel and that of the mineral apatites. The main variation that was found by some of these workers was that whereas pure hydroxyapatite contains only calcium and phosphorus anions, in enamel it was possible to find substitution of magnesium, chlorine, fluorine and sodium without any major alteration in the unit cell of the crystal.

Dallemagne and Melon[85, 86], after studying exhaustively the composition of bone salts, suggested that whereas bone and dentine consist primarily of calcium phosphate and calcium carbonate, enamel is composed of about 60% carbonate apatite, 30% α-tricalcium phosphate and 1% calcium carbonate and that the remainder consists of other minerals and organic matter. The carbonate apatite is in the enamel prisms whilst the free α-tricalcium phosphate is in the interprismatic substance.

The work of Kitchin[56], who employed polarised light to study the enamel, showed that the completely calcified enamel consists of submicroscopic crystallites of hexagonal shape. These are arranged within the enamel prisms with their long axes lying roughly parallel to the long axis of the prism. Thewlis[87] showed that the crystallites making up the enamel possess two angles of orientation, one being 5° to the prism axis and the other 40°. It is thought that the arrangement of the crystal-

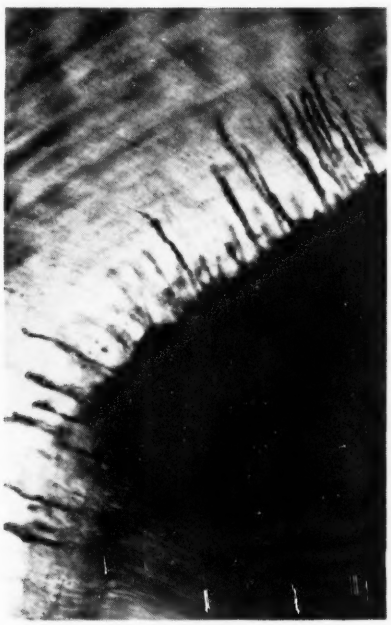

Fig. 9.—A longitudinal section of enamel showing enamel spindles arising from the dentino-enamel junction. (X 120. Transmitted light.)

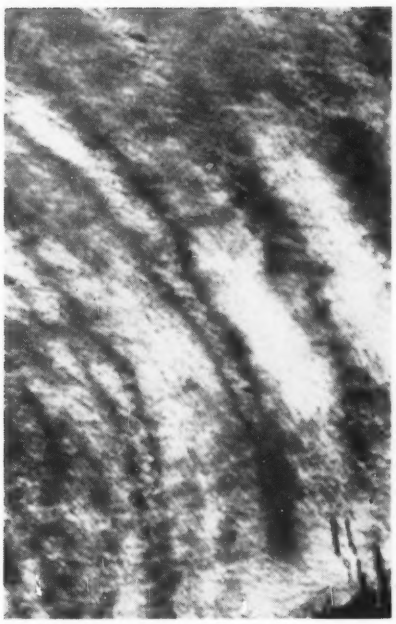

Fig. 11.—A longitudinal section of enamel, photographed by polarised light with parallel prisms, showing Hunter-Schreger lines in normal enamel. (X 175.)

Fig. 10.—Striae of Retzius seen in a longitudinal section of an incisor tooth. The striae can be seen curving upwards as they follow the contour of the tooth. (X 150. Transmitted light.)

Fig. 12.—A gold-shadowed celloidin replica of etched enamel in the area of the dentino-enamel junction. The variation in the direction of the enamel rods corresponding to the Hunter-Schreger lines is plainly seen. (X 120.)

lites in the interprismatic substance is not essentially different from the arrangement within the prisms, although Thewlis did say that the majority of crystals in the interprismatic substance have their axis at 40° to that of the prism.

Wyart and Tournay[88] examined the orientation of the apatite crystallites in enamel by using x-rays and concluded that the axes of the apatite prisms are contained within a cone of 30°. The axis of the cone makes an angle of 10° with the enamel surface towards the tip of the tooth. The apatite prisms appear to be independent of one another. They suggested, however, that their results gave only a statistical value.

The organic constituents.

At the beginning of this century, the belief that enamel was wholly inorganic was held by some of the foremost workers of the time. Williams[89] expressed the opinion that enamel was composed entirely of inorganic matter and described the opposing views of some of his contemporaries[90] as "vain imaginings." Support for Williams came from Tomes[7], and Hopewell-Smith[13, 14] also claimed enamel to be an inorganic, inert tissue.

However, as early as 1887, Thompson[91] had stated that "the salts of lime are deposited in an organic matrix of horny matter," and in 1906, Bodecker[1] very ably demonstrated the presence of an "organic matrix" by means of a celloidin decalcification method of preparing sections of enamel for microscopic examination. This brought about much discussion and eventually by 1923 the existence of organic matter in enamel was accepted.[92]

Because of the extremely tenuous nature of the organic matrix, estimates of its concentration in enamel have varied. Karlstrom[93] reported 0.3%, Sprawson and Bury[94, 95] 0.15%, Rosebury[33] 0.3-0.54%, Bowes and Murray[75] 0.97%, Le Fevre and Manly[96] 1.7% and Deakins and Volker[97] 1.46% (average).

The nature of the organic material, likewise, has not been readily ascertainable because of the difficulty of obtaining suitable samples for investigation. It was assumed that, because enamel is of ectodermal origin, the protein should bear some relationship to the keratins which are the least reactive of all proteins. Bodecker and Gies[34] were amongst the first to offer any histochemical proof of the presence and nature of protein matter in the enamel. They showed that a positive biuret test is given whilst the Molisch test for carbohydrates and the ammoniacal silver test for aldehydes are negative, indicating that the material is of a protein nature.[98] This conclusion was corroborated by the fact that a typical red colouration was obtained with Millon's reagent. The protein substance stains deeply with trypan blue, a dye which Gies had previously demonstrated could be absorbed into the enamel during its formation, but not after it had become fully calcified. His work was confirmed by Rosebury and Gies[99], who indicated that the protein has numerous characteristics similar to those of keratin. They believed that it is somewhat closely allied to the neurokeratins. They also stated that there appears to be neither soluble diffusible nor indiffusible protein in the enamel.

Chase[35] used histological staining to investigate the nature of the matrix and agreed that it is a protein, probably a keratin. Rosebury[33] re-stated that there is no soluble protein in enamel, and this view has been supported by Karshan and others[100] and Kanner.[101] It was further shown that enamel protein resists pepsin and trysin and gives a weak reaction for the presence of sulphur which made Rosebury consider that enamel protein resembles neurokeratin.

Pincus began an investigation of the nature of the organic material in 1935 and has published several papers since that time.[102-4] Whilst he found that the matrix possesses many properties in common with the keratins, its x-ray diffraction pattern differs from that of most keratins. He also found that it does not give a reaction for the presence of cystine and that it has a lower sulphur content than most keratins, there being no evidence of a sulphur-sulphur bond, the sulphur being bound only to carbon. His work indicated that tyrosine and methionine may be present. Wellings[105], however, later suggested that the tyrosine was an impurity. Kanner[101], by use of the polarograph, confirmed that cystine is absent.

Weinmann and others[106] showed that enamel which has just completed its maturation (as judged by conversion of the acid-insoluble matrix to acid-soluble, calcified enamel) has a protein content of about 4%. This value is much higher than that given for older teeth. Frisbie and others[32] assumed that the changes which occur in developing enamel continue after eruption—the process of progressive dehydration or desiccation eventually giving a protein which is more resistant to hydrolysis than is the protein in younger teeth.

Losee and Hess[107] carried out a series of investigations on enamel removed from noncarious, permanent molars. They demonstrated a variation in protein content in different types of teeth and claimed that cystine, methionine and phenylalanine are present in enamel protein. Histochemical reactions[108] pointed to the presence in the organic framework of the enamel of an acid mucopolysaccharide. Wislocki and Sognnaes also suggested that there

is no glycogen in the organic framework of the enamel.

Hutton and Nuckolls[109] were unable to demonstrate the presence of free amino acids in normal enamel but after hydrolysis they tentatively identified cystine and glutamic acid, glycine, aspartic acid, serine and tyrosine. Further, they indicated the presence of an unidentified carbohydrate which they thought may be a glycoprotein. Losee and others[110] suggested that the mol ratio of histidine, lysine and arginine (which they calculated to be 1:3:9) is in fairly close agreement with the ratio of 1:4:12 which has been suggested for a eukeratin.

Stack[111] suggests that enamel contains 0.4-0.8% of organic matter, including 0.1% of citric acid. Of the total organic matter he claims that some 7-9% is soluble protein.

Physical and Physiological Properties of Enamel.

Permeability and "vitality."

A question that arose quite early during the investigations into the structure and composition of enamel was whether the enamel is a truly "vital" tissue or is completely inert, and for many years there were two schools of thought as to whether enamel had any of the distinctive function of a living tissue or acted purely as a passive and protective covering. Black[112], in 1916, stated that:

> The tissues of the teeth are not changed in any way by physiological processes after they are once formed.

However, even in 1887, Thompson[91] had said that:

> It has been conclusively demonstrated that there are areas of living matter in the enamel, and that this living matter is in direct connection by an anastomosis more or less regular and continuous with the contents of the dentine tubulae. If this be true, then indeed there is osmosis by which nutrition is conveyed to the enamel however minute and inappreciable it may be.

The permeability of enamel, in a variety of conditions, was easily shown. Pickerill[113] placed teeth in a 10% solution of silver nitrate for 24 hours, exposed them to bright sunlight for a day and then washed them in salt water and prepared ground sections. By this means, he found that he was able to observe penetration of the silver nitrate to a depth varying between 0.13 mm. and 0.27 mm. He stated that, firstly, the amount of penetration bore a direct relationship to the structure of the tooth, depending upon its being "malacotic" or "sclerotic"; secondly, the degree of permeability of the enamel surface bore an inverse relationship to the length of time the tooth had been erupted; thirdly, the stain penetrates at first along the interprismatic substance and then seems to spread laterally into the prisms. He was not prepared to state specifically what materials are stained, but said:

> The fact remains that a fluid carrying a solid in solution can pass into the outer layers of enamel and that the solid is there fixed either by chemical combination or by precipitation.

Bunting and Rickert[114], and later Klein and Amberson[115], and Klein[116-7] demonstrated a dialytic flow of sodium and chloride ions through the enamel of extracted teeth. This was corroborated by an impressive mass of work by different investigators[39, 43, 47, 64, 118-122] using such dyes as alcoholic fuchsin, methyl blue, trypan blue and lithium carmine; they clearly demonstrated that it is possible to stain enamel by various means both *in voivo* and *in vitro*. They attempted to stain the enamel by penetrating both from the external surface and the dentino-enamel junction. They were able to do this and also showed that the more imperfect the enamel, the more deeply the stain penetrated. Jeffery[123] showed that the enamel is permeable to stain by using the method of staining by cataphoresis. The outer layers of enamel seem to be least permeable and there is an apparent variation as the post-eruptive tooth age becomes greater. A number of these workers stated that the penetration occurs through prism sheaths.

Entin[124] described a method of electro-endosmotic transport of water across enamel. Bodecker and Lefkowitz[63] stated that the bands of Schreger and rod sheaths are the main areas stained in enamel. They suggested the existence of an afferent and efferent flow of dental lymph in the dentinal tubules which are connected with the enamel. They agreed that the enamel is less permeable than the dentine and that its permeability varies with age. They also pointed out that in non-vital teeth the staining of enamel is far more uniform than in vital teeth and they assumed that a vital pulp exerts some influence on the dental tissues, one factor being the regulation of diffusion of dental lymph. Lefkowitz and Bodecker[64] claimed that stain can penetrate normal enamel from the dentino-enamel junction outwards but not from the external surface inwards. They compared the enamel to skin in having a protective function in not allowing the escape of lymph.

Berggren[125] carried out investigations, with the aid of radio-active isotopes, bacteria and bacterial toxins and dyes, to test the permeability of the enamel. Atkinson[126], using a method of cataphoresis, indicated that young enamel is permeable from both without and within to certain dyes when they are transported by means of an electric current. Under the same conditions old enamel appears to have an outer layer which, in parts, is impermeable to these dyes but which is permeable

to water. He also stated that the dye, whether transported by dialysis or cataphoresis, was always found in the prism sheath and further suggested the difference in staining between the old and the young enamel consists only in the reduction of the "pore-size" of the prism sheath.

A series of papers were published by Andresen[24] in which he claimed that it is possible, both by physiological and artificial means, to remineralise enamel. His reasoning was rather vague in many respects but it is obvious that he believed that enamel is a vital structure which not only is permeable but also can be subjected to ionic exchange. Weber[127] questioned the possibility of remineralisation in relation to the flow of salts from the pulp or saliva. He stated that where decalcification has been started, not only is the enamel not remineralised but that it will go on losing calcium ions to the saliva.

Chase[35] believed that as enamel forms in the developing tooth it undergoes a series of changes which he termed maturation and which are the analogues of the changes found in the various layers of the skin. These changes consist essentially of an increasing degree of keratinisation of the tissue with steady loss of solubility and reactivity. Evidence in support of this contention came from several investigators who indicated that the changes of maturation can continue even after the tooth has erupted. Fish[53] showed that the enamel of dogs' teeth is very permeable to methylene blue when a dog is young but it becomes less permeable, particularly on the outer surface, when the animal is older. He found some evidence of a similar phenomenon in human teeth.

Beust[46] suggested that most organic structures are calcified by the time the tooth has fully erupted:

> This renders those theories of resistance to caries that are based on the action of a buffer afforded by a lymph through the enamel paradoxical and no longer tenable.

Lura[128] favoured the idea of an enamel metabolism and stated that all the conditions for an exchange of the enzymic processes which form a regular metabolism are present in the hard tissues of the tooth.

Ostby[129] reviewed a large amount of literature in relation to this matter and suggested that the question of the vitality of hard structures of the teeth is decided by definitions. Those who believe that vitality is dependent upon the presence of phenomena characteristic of cell life naturally would regard the tooth structures as non-vital. However, those who consider the physical and chemical changes in the intercellular substance as representative of vital activities in the broader sense, will regard enamel as participating in the vital processes of the body.

Ostby's observations appear to be very pertinent for, indeed, most discussions concerning "vitality" have revolved around the interpretation of permeability. Widdowson[3] believed in the concept of a mere *physical* permeability where, although it may be possible to show the passage of different substances through the calcified tissue, there is nothing to indicate that it is a controlled interchange as we know it in other parts of the body. Such an opinion is held by many, including Enright, Friesell and Trescher[130], who stated:

> The results of an experiment carried out by Gies for a period of nearly ten years should convince us that there is no vital circulation in enamel.

However, if we favour the idea of a *physiological* permeability, it virtually implies that we believe the enamel protein is living organic matter capable of transporting and giving up ions even after the enamel has been fully developed. Studies in more recent years have added weight to the suggestion that some form of interchange may be possible within the enamel. In 1941, Wassermann and others[131] injected radio-active phosphorus into the blood stream of experimental animals and then demonstrated that it was taken up by the dental tissues including the enamel. The portal of entry was through the pulp or cemento-dentinal junction and there was no absorption from the saliva by the enamel in any of the experiments. Barnum and Armstrong[132] found that traces of radio-active phosphorus are able to penetrate the enamel surface. This was confirmed by Pederson and Schmidt-Neilsen.[133] However, both workers indicated that the amount is very small compared with that taken up by the dentine via the pulp.

In 1946, Forshufvud[134] injected haemolysed red corpuscles into exsanguinated dogs. He claimed that sections of the teeth showed staining in areas that corresponded to the Hunter-Schreger lines and suggested that this was evidence of a series of nutritional canals throughout the enamel. He then advanced the theory that there is yet another part of the circulatory system which he termed "the ultra-capillary system" which is made up of reticular fibres. He further suggested that the tufts, lamellae and prism sheaths which form the "enamel stroma" are part of this reticular, fibrous system.

Bartelstone and others[135] used a technique of injecting radio-active iodine into cats, then sacrificing them and making radio-autographs to determine the distribution of the iodine. The main disadvantage in this technique is that it does not lend itself to examination at any great degree of magnification.

Later Bartelstone[136] applied radio-active iodine to the external surface of intact enamel of cats' teeth and observed significant counts over the thyroid gland which indicated a penetration of radio-active iodine through the enamel and dentine into the blood stream.

Wainwright and Lemoine[137] demonstrated the rapid diffuse penetration of intact enamel and dentine by carbon[14]-labelled urea. These newer methods, using radio-active isotopes, have been well reviewed by Hevesy[138] who confirmed the existence of a slight but definite ionic interchange within formed enamel.

Whilst it is obvious that, because of the disappearance of the ameloblasts, it is impossible for any tissue repair in the ordinary sense to occur, we feel that we cannot subscribe to the assumption that chemical interchange within the formed enamel is impossible. It is quite possible that any ionic interchange or penetration of lymph that may occur in the enamel may be, to all intents and purposes, purely of a chemical and physical nature and even fortuitous, but in such a specialised tissue as enamel this could be important.

Micro-hardness.

It is obvious that such a highly calcified tissue as enamel will be extremely hard and it is known that enamel can resist the action of most abrasives quite easily. There is some doubt, however, as to whether there is any variation in hardness at the different levels of enamel. Hodge and McKay[139] showed a variation in the hardness at the different depths of enamel. Progressing from the dentine they obtained measurements (in Bierbaum hardness numbers) ranging from 330 to 910 through about nine-tenths of the thickness of enamel. In the extreme outer zone of both occlusal and buccal enamel they found a constant value of 2,050. Thewlis[83] confirmed this work by x-ray examination and suggested that there is a highly calcified layer of enamel about 0.1 mm. thick which invests the subjacent enamel like a skin. Hollander and Saper[140] denied the existence of this dense margin claiming that it was an artefact but Thewlis in 1937 confirmed his work to the satisfaction of most people.

Hodge[141] attempted to correlate micro-hardness of the enamel with the existence of carious lesions but he was unable to find any association at all. Applebaum[142], using Grenz rays, admitted the existence of a zone of hypercalcified enamel. He suggested that so opaque is it that even when early lesions occur in the enamel they do not remove sufficient calcium to change the Grenz ray picture.

Hinds[143] said that there is no difference in the hardness of enamel in erupted and non-erupted teeth. He attempted to explain the alleged variation in the hardness of the outer and inner layers by contending that in the process of maturation the enamel has to reduce its organic content and that it seemed that the enamel most remote from the surface would find most difficulty in getting rid of water and organic matter. This does not seem a particularly satisfactory explanation. Gustafson and Kling[144] carried out numerous investigations of the methods of testing micro-hardness and, whilst they did not deny the correctness of any of the results that have been previously published, they sounded a note of warning concerning the interpretation of many of the comparative results obtained. Earlier Gustafson[12] had suggested that the strong double refraction (which other workers previously had suggested is an indication of a high degree of mineralisation) which occurs at the dentino-enamel junction is due to the fact that the prisms in this area are all travelling in the same direction. He said that in the more superficial portions of the enamel the observations are more difficult because of the fact that the prisms in the various layers of the preparation run in different directions.

Swartz and Phillips[145] carried out a series of tests for hardness and solubility and showed that there was variation from tooth to tooth and that no correlation existed between the two.

Summary.

There are still many points to be clarified concerning the structure, composition and permeability of enamel. It would seem to be true to say that it is a highly inorganic structure. The calcium salts are disposed mainly in a microscopic arrangement of prisms which in turn have a submicroscopic crystalline structure. These crystallites have different axes of orientation. There is no definite agreement on the chemical nature of the inorganic salts, and if, as is believed, different salts are present, their distribution through the enamel substance has not been determined. The most widely accepted belief is that the prisms have a carbonate apatite structure with some calcium phosphate and calcium carbonate, whilst the material between the prisms is more of the nature of α-tricalcium phosphate. The organic material is scanty and is probably of two types. The main framework, which is represented by the prism sheaths in particular, is most likely similar to eukeratin whilst a ground substance is believed to be present also. Whether the ground substance is located only betweeen the prisms, or is spread throughout the entire structure as a hyaline matrix, is still to be decided. The enamel covering an erupted tooth is usually considered to be impermeable to larger molecules at least, but there is no doubt that it can be penetrated by such radicals as phosphate or iodide ions.

Structurally the enamel is composed of enamel prisms which are roughly hexagonal and pass in a wave-like course from the dentino-enamel junction to the surface of the enamel. They have cross striations, probably of an organic nature, and possess a submicroscopic, fibrillar network.

The prisms are separated from each other by a small but variable space in which area lies interprismatic substance. In the earlier writings a great amount of confusion arose because of the fact that "interprismatic substance" and "prism sheath" were considered to be identical and were therefore not treated as separate structures.

Other organic structures, namely tufts and lamellae, exist in the enamel and on occasions a lamella can extend throughout the depth of the enamel, forming a link between the external environment and the underlying dentine.

The enamel is originally covered by a primary and secondary enamel cuticle which is normally lost very soon after eruption.

Incremental lines can be seen in the enamel (known as striae of Retzius) and, under certain conditions, the variation in the direction of the enamel rods gives rise to an appearance of bands termed the Hunter-Schreger lines.

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Auto-Polymerising Acrylic Resin Restorations

A Review of the Present Status of these Materials.

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In discussing any filling materials used in Dentistry it should be remembered that although many gold inlays, porcelain inlays, amalgam and silicate restorations are successful, there are many failures. It is difficult to separate the failures due to faulty technique from those which might be attributed to lack of stability of the material, and condemnation of a material often comes both from the disregard and lack of understanding and proper control of the material. It is in an attempt to understand these acrylic materials better that some of their characteristic properties are considered.

Early attempts to polymerise unaltered acrylic monomer and polymer in prepared cavities were made by many investigators. Prominent amongst these in America was F. A. Slack[1], who reported some successes in 1943. However, the polymerisation time of these unmodified acrylic restorations was unreasonable and many failures resulted from the incomplete polymerisation.

During World War II modification of these acrylic materials in Germany resulted in a more satisfactory polymerisation time. Since then attempts have been made to improve the physical and chemical properties of these materials and some changes have been made in the types of activators used.

Chemical composition—effects of changes on materials.

The composition of auto-polymerising acrylic resins is generally:—

Polymer — essentially polymethyl methacrylate with benzoyl peroxide distributed.

Monomer—methyl methacrylate containing a slight amount of a tertiary amine—generally dimethyl para toluidine.[2]

Room temperature polymerisation has been achieved by the addition of a tertiary amine promoter to the monomer and benzoyl peroxide to the polymer. The addition of benzoyl peroxide to the polymer was well known but the use of a tertiary amine was a distinct advance made in Germany during World War II.

Another modification of the setting reaction is described by Salisbury[3] in which more desirable properties in the resulting polymer are produced through copolymerisation. Considerable alteration will occur if a dimonomer is incorporated with a simple monomer. A dimonomer is one which is difunctional: that is, it contains two polymerisable portions in its molecular structure. When this type of monomer polymerises, a cross-linked structure results.

More recently a different principle of activation has been described[4] using para toluene sulphinic acid. With this new activator it is claimed that some of the defects of the benzoyl peroxide, tertiary amine activated resins are overcome.

It was stressed by McLean[5] in 1950 that there is no direct acrylic filling material available that has superior properties to the standard processed resins; the only physical difference is the polymerisation time factor, coupled with a decreased volumetric shrinkage. The author feels sure that this principle still holds.

Properties.

(a) Polymerisation Time.

Polymerisation Time can be altered by:

1. Size of polymer particles—the smaller the particle size the shorter the polymerisation time.
2. Increase in temperature increases the rate of polymerisation.
3. Incorporation of a small amount of moisture may increase the rate of polymerisation. Using the sulphinic acid activated resins contamination with moisture has resulted in failure of the restorations.
4. Change in the amount and ratio of the activator and accelerator used.
5. Changes in type of activator used.
6. Variation of polymer-monomer ratio—thick mixes are somewhat faster than thin mixes.

(b) Temperature Rise during Polymerisation.

Polymerisation of present day direct resinous filling materials proceeds at room and mouth temperature with the evolution of heat.

The investigations of Wolcott, Paffenbarger and Schoonover[6] have shown that temperature rises of the smaller restorations are of no clinical significance. In general, faster curing and greater volumes produced higher peak temperatures.

(c) Thermal Conductivity and Coefficient of Expansion.

Acrylic resins are poor conductors of heat—this is a desirable property from the aspect of restoration of teeth. Skinner[2] states the thermal conductivity is approximately 120 times less than gold.

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The thermal coefficient of expansion for the resins is high by comparison with tooth enamel, amalgam, and gold. The thermal coefficients of expansion of various materials between 20°-50°C. are shown in Table I.

TABLE I.

THERMAL COEFFICIENT OF EXPANSION.[7]

Material.	*Coefficient.* (x10—6)
Tooth (root)	8.3
Tooth (across crown)	11.4
Tooth (root and crown)	7.8
Silicate cement	7.6
Amalgam	22 to 28
Gold	14.4
*Methyl-methacrylate resin	81

*While the figure above is the coefficient of thermal expansion for heat cured acrylic denture resin, it can be assumed that the self-curing resins have a coefficient thermal expansion of a similar order of magnitude.

It is considered that one of the physical properties of the acrylic resin filling materials which is of serious consequence is their high coefficient of thermal expansion.

Investigations of the fluid exchange between teeth and various restorative materials in these teeth have shown that this difference in coefficients of thermal expansion produces marginal percolation which may be an explanation for the recurrence of caries at the margins of some restorations.

(d) Hardness.

Describing one of the earlier self-curing acrylics in 1948 it was claimed that "large numbers of fillings tested over a trial period showed excellent resistance to attrition and staining."[9]

Actually acrylic resin is the softest of all the filling materials, being softer than pure gold, which is generally considered to be too weak and soft for inlay purposes. Hardness is further considered in relation to the indications and uses of acrylic resins (See Table II).

TABLE II.

COMPARATIVE HARDNESS OF DENTAL MATERIALS.[2]

Material.	*Knoop Hardness Number*
Tooth enamel	20
Tooth dentine	65
Silicate cement	70
Acrylic resin (inlay)	16
Acrylic resin (direct)	16
Pure gold	32
Amalgam	100

(e) Colour Stability.

A number of claims have been made about colour stability of acrylic resins. However, over the years most of the changes that have been made have been directed towards the improvement of colour stability. This would seem to indicate a definite weakness in this direction, and it has not yet been established that the more modern advances have resulted in definite colour stability.

In discussing the resins employing benzoyl peroxide—tertiary amine activation, McLean and Kramer[4] state as a defect their lack of colour stability. However, they do not present a clinical evaluation of the colour stability of the sulphinic acid activated resins, which are claimed by the manufacturers to be colour stable.

(f) Dimensional Changes during Polymerisation:

During polymerisation there is a shrinkage. In the construction of dentures, this shrinkage is controlled by:

(i) having the thickest mix of monomer and polymer practicable;

(ii) allowing polymerisation to proceed outside the flask before flask is packed;

(iii) building up and consolidating the pressure within the flask so that voids due to contraction are collapsed as they occur.

It appears that using the customary technique in the processing of denture base acrylics a reaction shrinkage of up to 1% is observed. Because of difficulties in duplicating these procedures in the placing of autopolymerising resins into prepared cavities, a greater polymerisation shrinkage must be expected. Skinner[2] has given the polymerisation shrinkage of the direct filling resins as from 5% to 8%.

It was in an attempt to overcome this reaction shrinkage that a non-pressure method of insertion of the materials was devised. The principle of the techinque suggested by Nealon[10] is based on the repeated replacement of volumetric loss. He says:

The volumetric loss can never be eliminated as long as it is necessary to use the monomer; however it can be compensated for.

Other techniques which are suggested are the so-called "stratified polymerisation technique"[4] and the use of an adhesive cavity seal. With these techniques the volumetric shrinkage is not reduced but the direction of the shrinkage is said to be controlled in such a way that marginal shrinkage is largely overcome.

(g) Water Sorption.

After insertion, the acrylic restorations are in contact with saliva and water sorption may take place. This water sorption is not great but may compensate for about half of the

shrinkage that takes place during polymerisation.

INDICATIONS AND USES

From a survey of the physical properties of acrylic resins, Tylman and Peyton[14] state:

> All synthetic resins are comparatively soft, so that as a substitute for gold or other filling materials it seems doubtful if the present acrylic resins can offer sufficient resistance to masticating forces and abrasion to be of use for all types of restoration.

It seems that the hardness of acrylic resin is sufficient only for restorations where the possibility of wear from scratching or abrasion is small, and for this reason the use of the material is usually restricted to proximal cavities on anterior teeth not involving the incisal angle and gingival third cavities. It may also be used for "facings," etc., where similar conditions apply.

Nealon[10] states:

> It is doubtful whether acrylics will ever replace silicate cements in the small proximal cavity where the pulp is protected by a heavy dentinal wall. The ease of manipulation of the silicates is in their favour.

METHODS OF MANIPULATION.

Cavity preparation:

Cavity preparation is similar to that used for silicate cements.

Colour matching:

With the tooth clean and moist and the shade guide moist a suitable shade or combination of shades is selected.

Insertion:

The cavity should be isolated and for this purpose the use of rubber dam is most helpful. The materials employing a sulphinic acid type activator must not be allowed to contact moisture before polymerisation has occurred. These materials are more susceptible to moisture contamination which may result in failure of the restoration.

Two main techniques are used for the insertion of self-curing resin restorations—the compression technique and the non-compression technique. Both techniques will be described in a general way but individual manufacturers' instructions must be followed if best results are to be obtained.

COMPRESSION TECHNIQUE.

With the compression technique an attempt is made with matrices and mechanical holding devices to consolidate the pressure within the cavity and collapse voids due to shrinkage as they occur. Considerable effort is required to obtain and maintain continuous pressure and it is recommended that a mechanical holding device be employed which will eliminate movement of the matrix band before polymerisation has taken place.

Anterior matrix retainers such as the Tofflemire (Fig. 1) which employ pre-formed stainless steel bands or a matrix holder such as the S.S. White matrix holder, which will take strip matrices of almost any material, are of great assistance. Many different types of matrix have been suggested but the more successful embody some mechanical holding device. The material for the bands should be metal (preferably stainless steel) or "cellophane," but not celluloid.

Mixing:

Acrylic monomer and polymer may be mixed on a slab or in a medicament glass—some evaporation of the monomer may be prevented by mixing in the latter; however, manufacturers' instructions should be followed closely.

Insertion:

The mix should be inserted before it becomes rubbery. The undercut areas should be filled with small pieces and then the cavity filled, if possible, with one large piece to avoid trapping air. The cavity should always be filled to excess.

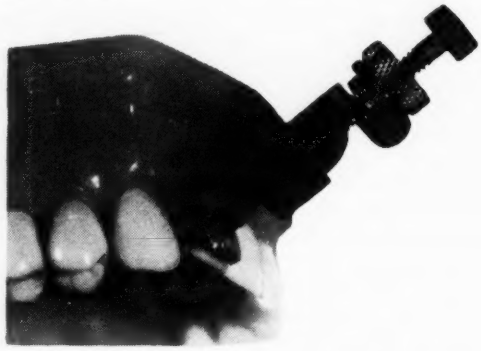

Fig. 1.—Anterior matrix retainer using pre-formed stainless steel bands (Tofflemire pattern).

In order to control more definitely the direction of the polymerisation shrinkage the so-called stratified polymerisation technique has been suggested. This technique involves the insertion of a fluid mix first over the walls and floor of the cavity, which is allowed to polymerise. A second mix is then inserted in the normal way and the volumetric shrinkage, though not eliminated, is said to be controlled so that marginal shrinkage is largely overcome. This control of shrinkage depends upon the first layer coming into intimate contact with the irregularities in the cavity walls and the second layer attaching to this.

NON-COMPRESSION TECHNIQUE.

Nealon[10] suggested a non-pressure method which uses the prepared cavity of the tooth as the site of conception and termination of polymerisation and takes advantage of the physical and chemical properties of the material.

The procedure described by Nealon is first of all to place 10 or 12 drops of monomer into a medicament glass. Polymer of the desired shade is placed in another glass which is warm and kept warm during the procedure by passing the medicament glass through a flame four or five times. The monomer and warm polymer are transferred in very small amounts to the prepared cavity using a No. 00 sable hair brush. The smaller and more frequent the additions, the greater the accuracy of marginal adaptation. The building-up period must take a minimum of five minutes for a medium size cavity such as a Class V (Black), and proportionately longer for larger cavities.

TRIMMING, FINISHING AND POLISHING.

With careful use of a matrix little excess should remain to be removed before the restoration is polished.

No trimming should be attempted until at least five minutes after removing the matrix and preferably should be limited at this stage to removal of excess with sharp cutting instruments. It is probably better to leave the restoration for 24 hours if the gross excess has been removed. Final trimming and polishing are carried out, bearing in mind that coarse abrasives should never be used and that heat should be avoided by using light intermittent pressure.

EFFECT OF SELF-CURING RESTORATIONS ON THE PULP.

Zander[12], Coy[13], Seelig[14], Kramer and McLean[15] have carried out investigations of the reaction of the dental pulp to self-curing acrylic restorations.

Both Zander and Seelig used experimental animals to test pulpal reaction. Zander[12] claimed that while some irritation was present, no single resin filling material caused a severe enough inflammatory reaction to label it as being severely irritating, and Seelig[14] concluded that the material itself produced no deleterious effect on the pulp. Both writers suggested that any irritation may be due to certain physical properties of the materials and also stressed the importance of the pulp condition at the time of insertion of the restoration.

As a result of his clinical study and experiments on animals, Coy[13] recommends a cement base over a lining of calcium hydroxide in deep seated cavities and no liner in cavities of normal depth. He agrees with Seelig and Zander that although there is a pathological pulpal response it is probably reversible.

McLean and Kramer[15], although they admit that their results are inconclusive, do suggest that the outcome of the inflammatory pulpal condition produced by the self-curing acrylic restorations is resolution.

On the other hand Grossman[16] recently conducted a survey in which he received reports from 76 dentists of the results of clinical use of the self-curing resins. From the results of this survey, it would appear that pulp death may occur under restorations of self-curing resin unless a cement base is used. (11,050 self-curing resin fillings were inserted by the dentists participating in this survey. As a control, the incidence of pulp deaths following the insertion of 9,086 silicate cement restorations for the same period was observed. In this series it was found that there were approximately four times as many pulp deaths after self-curing resin fillings were inserted as after silicate cements.) Grossman[16] definitely recomends the use of a cement base in all cases where a self-curing acrylic resin filling is to be used.

TABLE III.

COMPARISON WITH SILICATE CEMENT.

	Silicate Cement.	*Self-curing Acrylic.*
1. Cavity preparation:	Similar	
2. Cement base:	Required	? (may be required)
3. Manipulation time: (Mixing, insertion and matrix holding time)	Less	
4. Dimensional changes during setting or polymerisation:	Some shrinkage	Greater shrinkage
5. Colour matching:	Good	
6. Colour stability:	Known	?
7. Solubility in oral fluids:	Slight	Much less
8. Effect of dehydration:	Shrinkage (permanent)	Shrinkage (Compensated for on rehydration)
9. Coefficient of thermal expansion:	About the same as tooth structure	Much greater
10. Pulp reaction:	Known	?

CONCLUSION.

The main disadvantages of the auto-polymerising resins are the dimensional changes that occur during polymerisation and the coefficient of thermal expansion of the material.

Polymerisation shrinkage can be largely controlled, but the high coefficient of thermal expansion may result in marginal percolation of mouth fluids. Whether this marginal percolation is of clinical significance can only be determined by careful observation over an extended period.

The effect of these materials on the dental pulp is as yet undecided but the use of an inert material would prevent possible pulp irritation from the auto-polymerisation acrylic resins.

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Cancer: Retrospect and Prospect*

Charles S. Cameron, M.D.†

Within slightly more than a generation, cancer in the United States has risen from eighth place to second among the causes of death, an increase so striking as to have called forth greatly increased efforts to discover the cause, or rather the causes, of this widespread scourge. These research endeavours include a growing interest in cancer's epidemiology—the investigation of the pattern of its occurrence among various cultural, national, geographical, and occupational groups, a type of study which has heretofore been limited and, indeed, haphazard. There have appeared numerous reports of the rarity of cancer among Eskimos, native tribesmen of the Belgian Congo, and other more or less primitive societies, but, with all respect due the explorers, missionaries and isolated doctors who are the usual sources of such data, they have been unsupported by adequate statistical control and analyses as a rule.

The record of the incidence and prevalence of cancer throughout the world is largely blank and in fewer than a half dozen countries do such figures begin to approach reliability. The United States is not among them, for, while some system of cancer case-reporting is provided in over half our states, in only a few states are the systems designed to obtain complete reporting. In respect of mortality records, the picture is much brighter, although, again, there is enormous variation throughout the world in the degree of their accuracy and completeness.

While we may agree that cancer is a world problem, it is apparent that it is not the same problem everywhere. The 1940 figures place Ceylon in the lowest position among 29 countries (Fig. 1) with 11.3 cancer deaths per 100,000 of population; Switzerland leads with 176 cancer deaths per 100,000, so that the rate of deaths from cancer among the Swiss is fifteen times that of the inhabitants of Ceylon. The United States stands thirteenth in the list, with 120 cancer deaths per 100,000 of population. (The rate for 1946 in the United States was 130.) A map adaptation of these figures (Fig. 2) provides a rough index of the extent of the cancer problem and one which remains substantially unchanged when examined from different points of view. First, the map discloses a parallelism of sorts between the recorded cancer death rates and the general cultural level as established by such factors as literacy, standards of living, medical care, and industrial technology. If these and similar aspects of living be accepted as indices of "civilization," it might be argued *at this point* that cancer is a disease of civilization.

The ability to organize for cultural effort is obviously easier for compact units like cities and so it is in the city that we should expect to find higher cancer death rates. This appears to be true for at least our own

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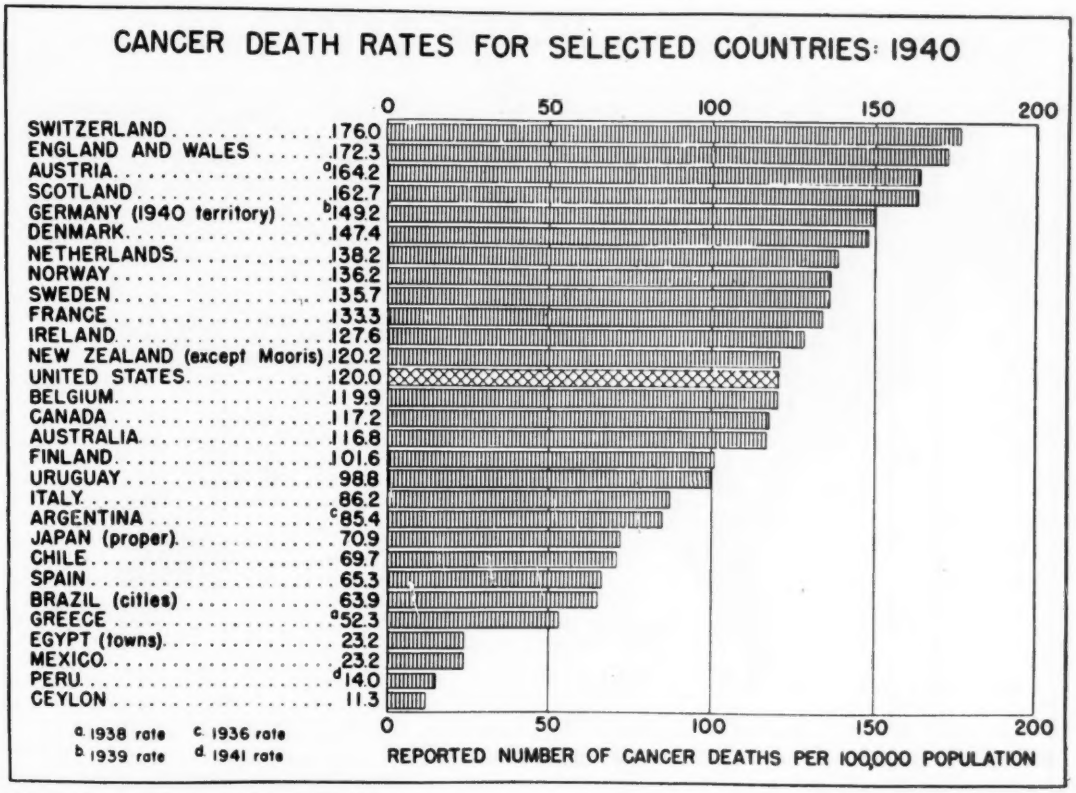

Source of data: Summary of International Vital Statistics, 1937-1944.

Fig. 1

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4-48 American Cancer Society

country. (Fig. 3.) A rather striking correlation between the degree of urbanization and the cancer death rate exists, indicating that states having the most people living in cities have the highest cancer death rates, the probable reasons for which will appear later.

A second parallelism is found when we compare cancer death rates with the death rates from all causes (Table 1). Countries having high general death rates tend to have low specific rates for cancer, whereas countries having low general death rates have high cancer rates. A low general death rate is the resultant of such vectors as high per capita wealth, high literacy rate, superior living standards with emphasis on nutrition and high indices of medical practice—both preventive, in the broad sense of public sanitation, and therapeutic, in the sense of adequate facilities and competent service. When these factors operate, neonatal deaths and those peculiar to infancy decline; deaths from the epidemic diseases of childhood fall off; deaths from tuberculosis, yellow fever, and from the

TABLE I

DEATH RATES[1], ALL CAUSES AND CANCER BY COUNTRY
SELECTED COUNTRIES — 1940

Country.	Total Rate.	Cancer Rate.	Country.	Total Rate.	Cancer Rate.
Switzerland	1,201.0	176.0	Egypt	3,034.1	23.2
Argentina[2]	1,195.2	85.4	Mexico	2,335.0	23.2
Germany[3]	1,168.0	146.9	Guatemala[4]	2,146.0	11.4
Sweden	1,141.8	135.7	Chile	2,145.3	69.7
Norway	1,088.0	136.2	Finland	1,895.9	101.6
United States	1,074.1	120.0	France	1,886.9	133.3
Denmark	1,033.5	147.4	Rumania[5]	1,857.6	43.8
Iceland	990.4	126.3	Brazil	1,831.2	63.9
Netherlands	988.0	138.2	El Salvador	1,747.4	12.9
Canada	976.1	117.2	Costa Rica	1,708.7	62.8
Australia	972.4	116.8	Venezuela	1,659.8	25.8
Uruguay	960.3	98.8	Japan	1,652.4	70.9
U. of S. Africa	940.3	104.7	Spain	1,641.9	65.3
New Zealand	923.6	120.2	Belgium	1,612.1	119.9

[1]Rate per 100,000 population; [2]1936 rate; [3]1938 rate; [4]1943 rate; [5]1939 rate.
(From: Summary of International Vital Statistics, 1937-1944.)

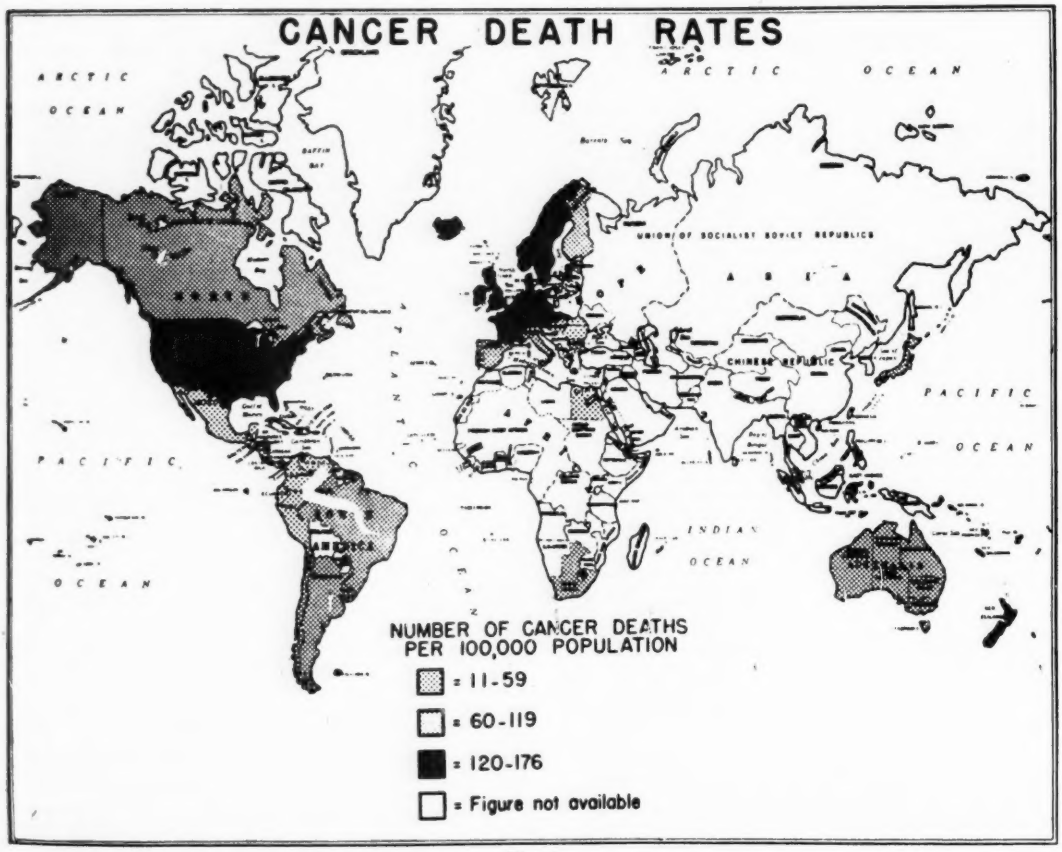


Fig. 2

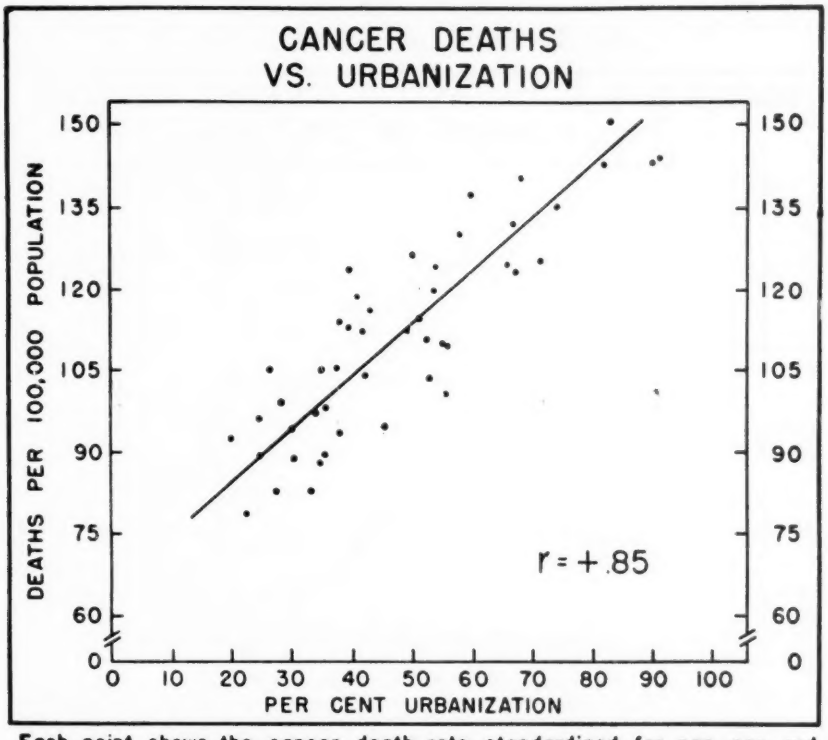
sequels of malaria and hookworm disease are reduced; deaths from typhoid and smallpox become disgraces to communities. Thus, in countries with low general death rates it becomes the happy custom to live on to forty and beyond, and then to die of the only things left to die from—cancer or heart disease.

Adequate facilities and competent medical care have been cited as factors producing low general death rates and high cancer death rates. This may be demonstrated by comparing the number of doctors per unit of population with the death rates from infectious diseases on the one hand and with the death rates from cancer on the other. Due to the widespread dislocations which have resulted from the war, it has not been possible to obtain reliable figures for most countries, and only those derived from our own study in this country are presented. In figure 4 each dot represents the death rate for infectious and parasitic diseases *and* the number of doctors per 100,000 of population in each of the forty-eight states. An inverse ratio exists, namely, the greater the number of doctors, the fewer the deaths from infectious and parasitic diseases. However, for cancer (Fig. 5) the dots follow the opposite direction, establishing a direct proportion, that is, the greater the number of doctors per unit of population, the greater the number of deaths from cancer. This paradox could lead to this odd conclusion: If you have cancer, do not go near a doctor. But, what it probably means is that in areas well supplied with doctors, fewer people die from cancer unattended, undiagnosed, and untreated.

There is another explanation for the higher death rate in cities: Large cities, with large and reputable medical centres, draw in respectable numbers of outlanders suffering from cancer. Their deaths are generally recorded in the mortality statistics of the city in which they die.

Low general death rates are synonymous with increased life expectancy or longevity. And, as has been noted, increased longevity of a population is attended by an increase in its cancer death rate. The cancer death rate rises appreciably for males at about 40, and for females at about 35. Therefore, the larger the segment of a population aged 40 and over,

the greater will be the cancer death rate. Our pattern of cancer death rates for the world corresponds fairly well with the pattern of population age, and, in general, those countries with high death rates due to cancer are seen to have older populations (Fig. 6). In 1900, not quite one-quarter of our people were 40 or over. In 1940, one-third of the population was reaching the 40-year mark, and in fifty years or so, almost one-half of all persons living in the United States will be oldsters, if the present trend continues and begins to stabilize. Therefore, if it is held that cancer is not a world problem today, it will become so, as the benefits of preventive medicine and adequate medical services are made increasingly accessible.



Each point shows the cancer death rate standardized for age, sex, and race and the per cent urbanization for one of the 48 states: 1939-1941.

SOURCE OF DATA: VITAL STATISTICS OF THE UNITED STATES

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8-47

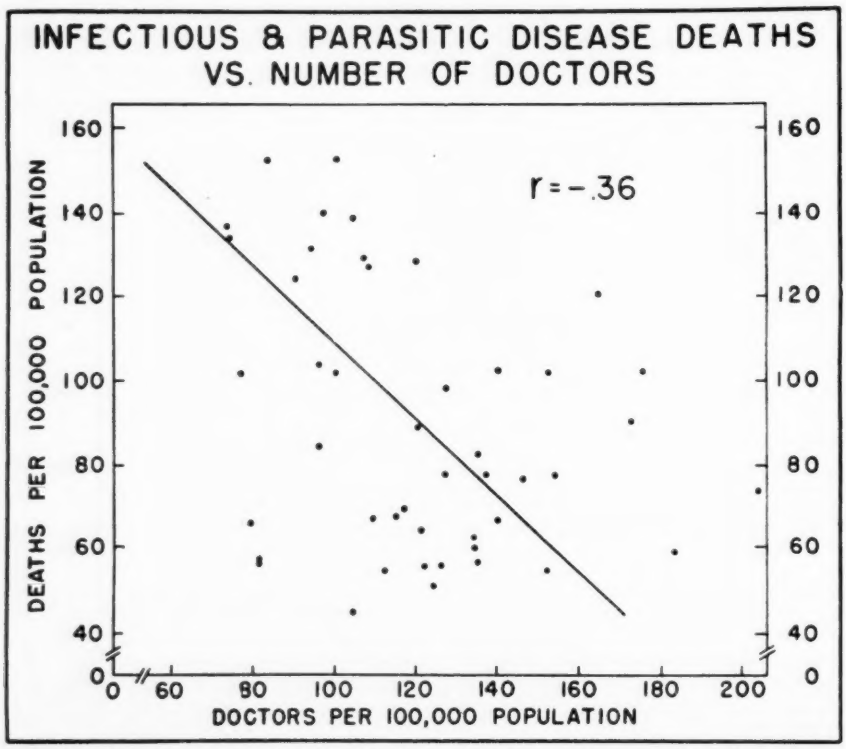
Fig. 3

The conclusions to be drawn from the foregoing remarks are: (1) wide variations exist in the recorded death rates from cancer from forty-six nations of the world; (2) no satisfactory biological explanation for these differences, other than length of life, has been offered; (3) valid explanations for these differences are found in the varying degrees of completeness in reporting deaths from cancer, varying degrees of accuracy of diagnosis when deaths are so reported, and wide variations in age composition, wealth, literacy, living standards and standards of medical care existing among the units studied; (4) there is little reason to believe that people living in a country reporting a low cancer death rate are generally less susceptible to cancer than inhabitants of countries reporting high cancer death rates; (5) low cancer death rates suggest incomplete statistical coverage; (6) more general, complete, and accurate reporting, not only of deaths, but of the incidence of cancer throughout the world will be necessary before more dependable conclusions regarding cancer's ecology can be reached.

While the observed differences in the cancer death rates as reported in various parts of the world are difficult of interpretation, the ground is somewhat surer in respect of recorded differences in frequency of site-specific cancers. For example, suppose that one registration area reports that the death rate from cancer of the stomach in white female patients considerably exceeds the gastric cancer death rate from Negro female patients. We might

reason that this is because, in the study area, Negro female patients are underprivileged, do not seek medical care as often as white patients, and do not get as good attention when they do seek it. Yet if that same area reports that the death rate from cancer of the uterus is respectably higher in Negro female patients than in white female patients, the "underprivileged" explanation will not stand. Again, if two widely separated registration areas reporting general death rates and cancer death rates of the same order of completeness or incompleteness of total registration, for we are dealing not with the indeterminate factor of how many cases are unreported, but with known quantities within a series; and, if the series is large enough, the data are meaningful.

There are certain countries of the world, or, better, certain geographical areas of the world, where there are remarkably high rates for certain anatomical varieties of cancer. The predilection of cancer for particular organs or parts in such areas can, in a num-



Each point shows the death rate for infectious and parasitic diseases and the relative number of doctors for one of the 48 states, 1939-1941.

SOURCE OF DATA: VITAL STATISTICS OF THE UNITED STATES

AMERICAN CANCER SOCIETY
STATISTICS DEPARTMENT
8-47

Fig. 4

magnitude, and having comparable medical services, wealth, and literacy, report significantly different death or incidence rates for cancer of the same site, we have a finding of importance. An example of this is found in comparison of the incidence of cancer of the stomach among white males in Denmark and New York State. The rate for Denmark is 23 per cent. of all new cancers among male patients, while the corresponding figure for New York State is only 10 per cent. It may not be unfair to say that site-specific rate differences have significance regardless of the ber of instances, be satisfactorily explained on a simple cause-effect basis, that is, the causal genesis of certain cancers can be identified in habits of the people, social customs, religious practices, occupation, endemic parasitic infections, climate, and geological influences. Tumors resulting from such circumstances are often referred to as environmental. The relation between cancer of the buccal mucosa and betel chewing is adequately documented. The betel habit is widespread in certain parts of India, Ceylon, and the Philippines. In reported series including cancers of

all types from Southern India, Travancore, Madras, and Ceylon, cancer of the buccal cavity accounted for 91 per cent., 38 per cent., 42 per cent., and 32 per cent., respectively, of all cancer observed. While oral cancer is rare among white women, it accounts for 12 per cent. of all cancer in Filipino women, so that the sex ratio of oral cancer among Filipinos is inverse to that found among white persons in the temperate zone. Squamous carcinoma of the skin of the abdominal wall is seen among the shepherds of Kashmir in Northern India who keep warm by means of the kangri, an earthenware bowl contained in a basket and fastened to the abdomen; the bowl is filled with wood embers sprinkled with water to prevent active combustion. Within fifty years, Neve noted more than 2,000 such cancers in Kashmir. At least one element of climate is involved in cancer, namely, sunlight. In our own southwest and in Argentina, the rate of cancer of the skin among light-complexioned inhabitants is significantly higher than in areas where longer winters discourage year-round occupational and recreational exposure to the sun.

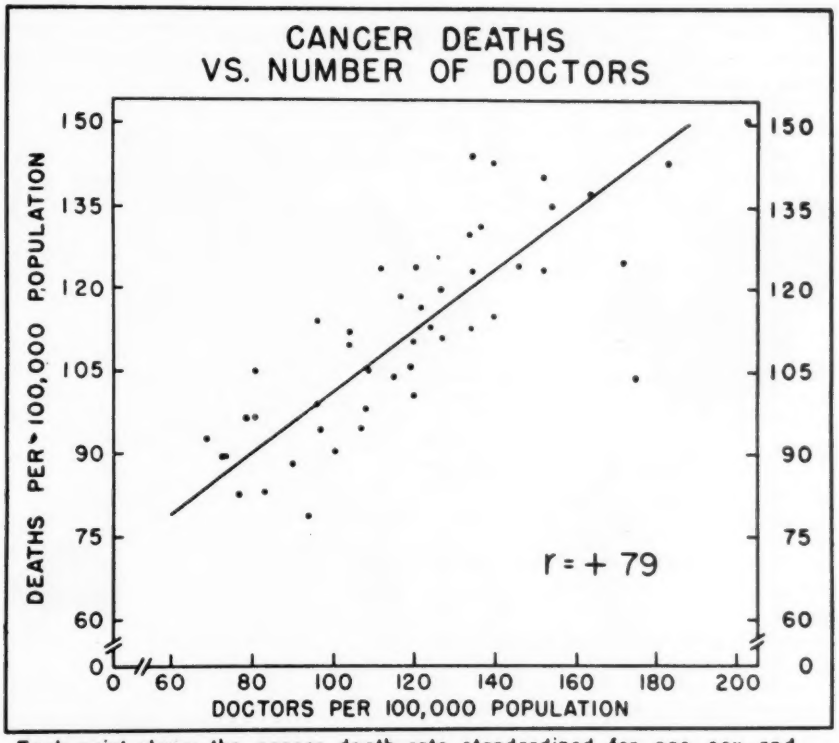


Fig. 5

The practice of circumcision, rooted in Hebrew and Mohammedan dogma, has rendered Jews immune from cancer of the penis, which accounts for 15 per cent. of all cancer in Chinese males and is probably equally common in the Siamese. Penile cancer is also said to be rare among Mohammedans, who also practise circumcision at puberty. Other data indicate that this form of cancer is more common among Mohammedans than among Jews, which, if true, may be related to the older age at which the rite is performed among the Mohammedans. It would be interesting to speculate as to whether any aspect of orthodox ritual can be made to account for the well-established low incidence of uterine cancer among Jewish women. It has even been suggested that uncircumcised males "give" cancer of the cervix to their wives.

An example of endemic parasitic infection as a formal cause of cancer is seen in primary cancer of the liver associated with helminthic infestation among inhabitants of oriental and tropical countries. Hueper[1] has summarized the observations from numerous reliable sources and finds that primary carcinoma of

the liver is much more frequent in African Negroes, Asiatic, Javanese and Chinese than among members of the white race living in tropical or moderate climates, and supports that statement by pointing out that 29 per cent. of 359 cancers in African Negroes, 57 per cent. of all cancers of all Javanese, and 30.8 per cent. of all cancers in Chinese were primary liver carcinomas, whereas only 1 per cent. to 5 per cent. of all cancers among white patients are primary in this organ. Numerous investigators have firmly established the relationship of schistosomiasis to vesical neoplasms, so prevalent in Egypt, the Sudan, Morocco, Somaliland, Madagascar, and the Transvaal, and in Syria, Palestine, and India. It has been stated that 70 per cent. to 90 per cent. of the population of Egypt is infected with schistosoma; however, the great majority of infected persons do not develop vesical tumors. The incidence of vesical cancer in vesical Bilharzia infection is said to be one case of cancer in 1,600 cases of clinical schistosomiasis. This figure is probably low, for the frequency of bladder cancer associated with Bilharziasis is many times higher in data based on autopsy findings in persons dying from parasitic infection. Twenty per cent. of all cancers in one reported series were Bilharzia cancers of the bladder; in Europe and the United States, cancer of the bladder accounts for 2 per cent. to 3 per cent. of all cancer.

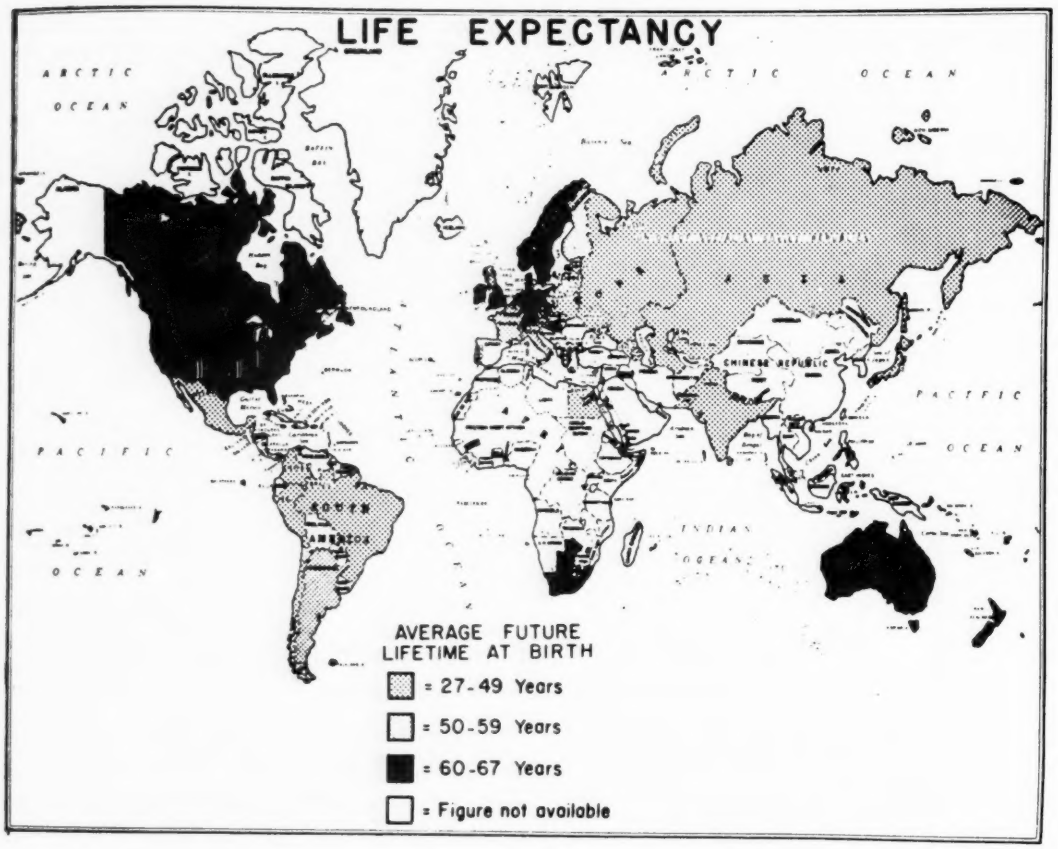


Fig. 6

Geological influences have been indicted as contributing to high rates of certain types of cancer in specific areas. Water rich in arsenic-containing ores has been related to relatively high skin cancer rates in Silesia and parts of Argentina. Carcinoma of the thyroid is ten times more frequent among the Swiss than in goitre-free regions of the United States, a fact directly attributable to low iodine content in the soil and water of Switzerland. In regions with endemic goitre, from 2.5 per cent. to 4 per cent. of all cancers are found in the thyroid, while in non-goitre areas, the relative frequency of thyroid cancer is only from 0.4 per cent. to 0.5 per cent.

The relationship of modern industry to cancer is receiving increasing attention, and the list of environmental agents to which workers in many industries are exposed and which are more or less well attested carcinogens is impressive. Hueper[1] has pointed out that the chemical industry has excelled in the creation of artificial environment in its production of dyes, mordants, explosives, plastics, insecticides, solvents, resins, lacquers, pigments, paints, fuels, lubricants, refrigerants, radioactive substances, and all this in the chemical phases of industry alone. Although occupational cancer is not specifically a function of geography, the increased frequency of certain kinds of cancer attending certain industrial operations must in a definite, though perhaps small, degree contribute to the parallelism we have previously pointed out between the general cancer incidence or death rate and the industrialization of a nation.

TABLE II

Death Rate per 100,000 Population in 13 Countries Cancer of the Stomach, Liver, and Esophagus			
Switzerland	70.4	Uruguay	35.6
Holland	62.2	England-Wales	31.4
Norway	61.4	Ireland	31.0
Bavaria	59.4	United States	28.3
Japan	40.0	Australia	27.4
Scotland	36.0	Italy	26.2
		Cuba	12.7

Death Rate per 100,000 Population in 11 Countries Cancer of the Skin			
United States	2.7	Switzerland	1.9
Ireland	2.7	Scotland	1.7
Australia	2.3	Holland	1.4
England-Wales	2.1	Uruguay	1.1
Cuba	2.0	Bavaria	0.8
		Japan	0.7

(From: Hoffman—The Mortality from Cancer Throughout the World, Prudential Press.)

Over and above these variations in organ-incidence of cancer in different areas, which can be explained in some measure, there exist differences in incidence and mortality rates for specific sites, for which we have today no basis or orientation. It has previously been mentioned that cancer of the stomach among men in Denmark is over twice as common as it is among the men of New York State, two areas selected for comparison because of generally comparable conditions. Some years ago, Hoffman[2] compared the relative frequency of cancer of the stomach, liver and esophagus combined in thirteen countries. (Table II.) The relative mortality for cancer in these three sites was higher in Uruguay and Japan than in the United States registration area and in England, and, further, the rate was twice as high in Switzerland, the Netherlands and Norway as in the United States. It is improbable that the practice of medicine or the accuracy of death certification is so superior in Uruguay, Japan, Norway, Holland, and Switzerland, as to account for all of this disparity. The significance of these figures becomes greater when they are compared with the table of frequency of cancer of the skin covering eleven countries. The highest rates are found for the United States, Ireland, Australia and England—with Japan, Uruguay, Holland, and Switzerland being low. These tables suggest two possibilities: First, in respect of diagnosis, Switzerland, Holland, Uruguay and Japan occupy unfavourable positions as regards accessible cancer, but decidedly favourable ones as regards efficiency in the diagnosis of the inaccessible forms of cancer—of the stomach, liver, and esophagus; and, second, the observed differences are valid, and indicate country-specific susceptibility to cancer of the skin on one hand and to cancer of the stomach, liver, and esophagus on the other. Of the two, the latter appears more reasonable. Similar disparity is seen in the mortality rates for the two forms of cancer so common in women, namely, cancer of the breast and cancer of the generative organs (Table III). The highest rate for cancer of the female generative organs in the group of countries represented here is shown to prevail in England, followed by the United States. The lowest rates occurred in Norway, Uruguay, and Ireland. In the comparative table for cancer of the stomach, liver, and esophagus, the rate for Norway was third highest, whereas for cancer of the female genital tract, Norway is lowest of the thirteen countries represented. The highest mortality rate for cancer of the female breast among the thirteen countries compared here occurred in England, followed by Scotland, Ireland, Switzerland, and the United States. The lowest rates were found in Japan, Uruguay, Cuba, Italy, and Norway. It is curious that deaths from cancer of the female generative organs were recorded almost as often in Japan as in England, while deaths from cancer of the breast are recorded ten times as often in England as in Japan; and it becomes the more

TABLE III

Rate per 100,000 Female Population in 13 Countries Deaths of Cancer of the Female Genital Organs			
England-Wales	24.2	Cuba	18.9
United States	22.1	Italy	16.0
Bavaria	21.6	Australia	15.5
Switzerland	21.4	Holland	13.2
Japan	20.9	Ireland	12.8
Scotland	20.6	Uruguay	12.2
		Norway	11.5

Rate per 100,000 Female Population in 13 Countries Deaths of Cancer of the Female Breast			
England-Wales	17.9	Holland	9.6
Scotland	15.4	Bavaria	9.1
Ireland	14.0	Norway	7.3
Switzerland	13.6	Italy	5.8
United States	13.3	Cuba	4.5
Australia	10.6	Uruguay	3.7
		Japan	1.8

(From: Hoffman—The Mortality from Cancer Throughout the World, Prudential Press.)

curious when we consider the relative accessibility of the two forms. Hoffman was led to the conclusion that the local variations in cancer frequency throughout the world were primarily conditioned by local causes rather than by faulty diagnosis or defective methods of death registration.

It is clear that "local causes" are of importance in determining the pattern of cancer throughout the world. While the foregoing observations have been oriented to statistical operations involving large numbers of people, the ultimate goal of such biometrical studies is, like that of the physician, to prevent illness or death. For a number of varieties of cancer, proximate causes are sufficiently proved to justify broad prophylactic measures. For others, the evidence is as yet presumptive or merely suspicious; more and better epidemiological investigations need to be undertaken in order to confirm or deny questionable environmental factors. The immediate dividends of investigations of this sort, in terms of lives saved and illness prevented, is obviously very great. Of hardly less value is their identification of problems calling for fundamental laboratory research. It is possible that serious attention to these epidemiologic data on the part of investigators in the basic scientific disciplines would shorten the research task ahead appreciably.

Genetics.

The hereditary origin of cancer has long been a widely held vulgar credo. Among scientists, opinions regarding its importance have varied with the mental attitude and experience of the observer. In recent years much evidence has been gathered which seems to show that heredity is the most important and determining factor in the incidence of some forms of tumors, especially in the lower animals, and that its influence remains undemonstrated in most of the forms of human cancer. Human tumors having heritable features are seen in retinal glioma, intestinal polyposis and neurofibromatosis.

The earliest observations concerned cancer families. In 1837 Warren[3] reported a family history in which the grandfather had cancer of the lip, while his son, his daughter, two sisters and a niece all died of cancer of the breast. Perhaps the best documented of these older reports is that of Warthin[4], giving a record of seventeen instances of cancer, chiefly of the uterus or stomach, among forty-eight descendants of a cancerous great-grandfather. Sibley[5] observed cancer of the breast in a mother and her five daughters—and in the left breast of all of them.

The occurrence of tumors in identical twins is a fairly convincing illustration of the influence of heredity. McFarland and Mead[6] collected forty instances of such tumors in twenty pairs of twins.

In respect of cancer in general, without regard to site, the very numerous statistical studies of families of cancer patients appear to line up about evenly on each side of the question of heritable susceptibility.

The recent work of Macklin[7] deserves fresh interest: Careful studies of the family histories of women with cancer of the breast disclosed that breast cancer was significantly more common among the relatives of breast cancer patients than in the general population.

Many experiments in laboratory genetics appeared to all but remove the heritable nature of certain kinds of animal cancer from its realm of hypothesis. As early as 1911, it had been observed that mice with a cancerous ancestry were more likely to develop malignant tumors than animals without such a background.

The monumental studies of Maude Slye led her to conclude that: (1) the inheritance behaviour of neoplasms is that of Mendelian recessive; (2) double cancer parentage yields 100 per cent. tumor strains; (3) single cancer parentage yields heterozygotes in the first hybrid generation. These, whether inbred or hybridized with other heterozygotes, yield, in the second hybrid generation, non-cancerous, heterozygous and cancerous progeny in the proportion of 1:2 :1; (4) the mating of a cancerous with a heterozygous individual gives approximately 50 per cent. cancerous and 50 per cent. heterozygous offspring; (5) double non-cancerous parentage yields 100 per cent. non-cancerous strains; (6) immunity to cancer is also inherited, as a dominant; (7) two factors are necessary to produce cancer: inherited susceptibility and particular forms of chronic irritation.

These conclusions were early modified by other geneticists for whom the Mendelian postulates were inadequate explanations of the phenomena observed; for example, it has been suggested that several Mendelizing factors are required to account for a given character, including the cancer tendency.

Milk Factor.

The inadequacy of the unitarian genetic hypothesis was further shown when the milk factor was demonstrated by Bittner[8] in 1936. That some maternal or extrachromosomal influence was involved in mammary cancer in the mouse was strongly suggested by the observations resulting from several reciprocal cross-breeding experiments with strains having high and low incidences of spontaneous

mammary cancer. In every cross, the hybrids with maternal parents belonging to cancerous strains showed a high rate of occurrence, whereas only a few tumors appeared in the reciprocal hybrids—fathers of high cancer strains, mothers from low. These findings pointed toward an extrachromosomal, maternal influence including, among several possibilities, a milk-borne breast cancer inciter.

During the intervening years, the mammary tumor agent has been investigated by various methods. Electron micrographic photographs of material obtained from the stomachs of suckling offspring of a strain of female mice noted for its high mammary cancer rate show shadow-casting spheroidal particles which conform to our present data bearing on virus morphology, and Gross and his associates[9] have found similar structures in breast milk of women whose female siblings or antecedents had had cancer of the breast. (Fig. 7).

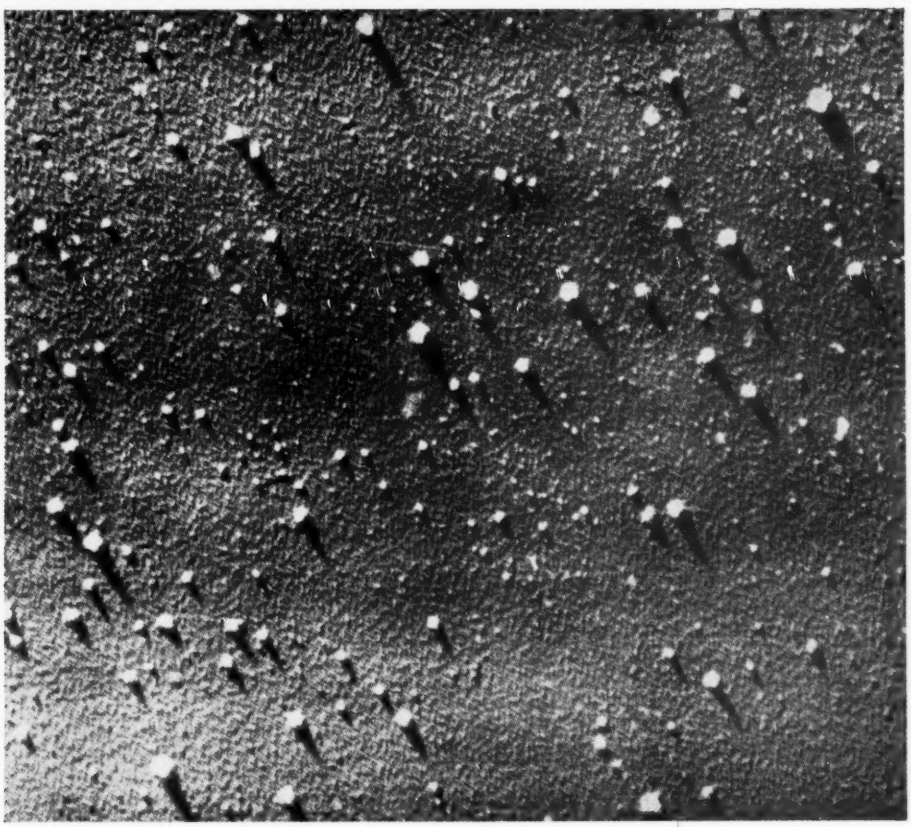


Fig. 7—Electron micrograph of a milk sample from a healthy nursing woman whose sister had cancer of the breast. Numerous spherical particles present, frequently arranged in pairs or clusters. Chromium shadowed. (X10,000.) (From Gross, Gessler, and McCarty: Proc. Soc. Exper. Biol. & Med. 270-276, 1950.)

The question arises, "Have these observations significance for human cancer?" Macklin's data, as I offered it a few minutes ago, would provide affirmation, but I withheld that portion of her analysis which is more critical for the question of a human milk factor, namely, the comparison of the incidence of cancer among the female antecedents on the paternal side with those on the mother's side. Cancer of the breast was, in fact, slightly more common among the females related to breast cancer patients on the father's side than among those related through the mother. These data do not lend support to the importance of the milk factor in human breast cancer.

Whatever the implication in all this, the milk factor has revived serious attention to virology in relation to neoplasms. Viruses had previously been assumed to be the causative agent in fowl sarcoma, rabbit papilloma, adenocarcinoma of the frog kidney, and, recently, mouse endothelioma.

A recent experiment by Gross[10] is suggestive of similarity in one respect between mouse

mammary cancer and mouse leukemia (Fig. 8). In the AK inbred strain, leukemia occurs spontaneously in 70 per cent. of the males and females. Inoculation of leukemia cell suspensions obtained from mice with leukemia will not usually induce leukemia in individuals of other lines, although it will do so regularly in mice of the leukemia-susceptible line. Specifically, as attempted in the past, AK leukemia could rarely be transferred to C_3H or other resistant strains. When, however, leukemic cell suspensions from AK mice were injected into C_3H infants, it was found that it could be transferred and that the younger the mouse, the greater were the chances of successful transfer. Thus, by taking advantage of the critical factor of time, leukemia could be transferred with

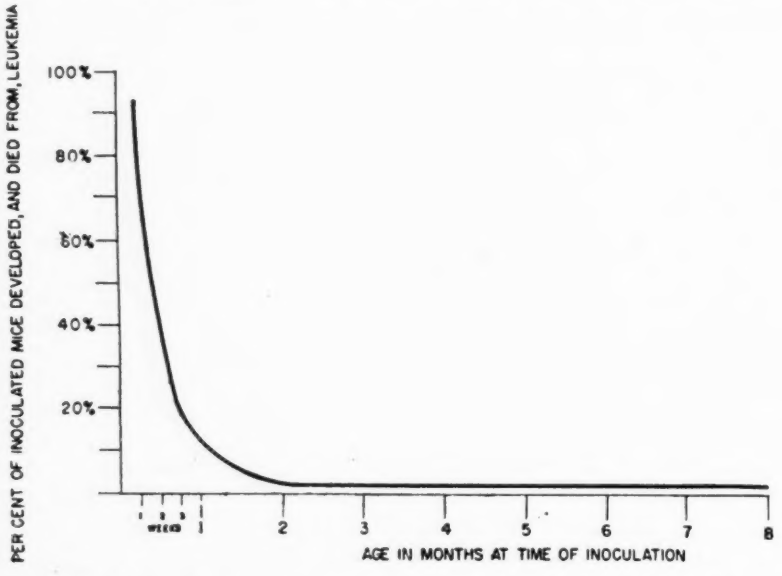


Fig. 8—Rate of "takes" of leukemia following injection of mouse leukemic cell inoculum into mice of resistant strain. (From Gross, L: Proc. Soc. Exper. Biol. & Med. 246-248).

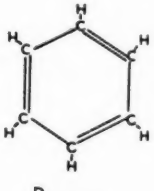
nearly regularity to a strain previously believed resistant. The disease does not usually appear until after the lapse of a latent period, and this feature is characteristic of certain viruses.

Chemical Carcinogenesis.

The study of chemical carcinogenesis has, I think, the most venerable history of any of the modern approaches to the essential nature of cancer. The linking of coal soot to scrotal cancer by Pott in 1775, the association of paraffin exposure and scrotal cancer by Von Volkman one hundred years later, and the crucial experiment by Yamagiwa in 1913, by which he produced cancer on the ears of rabbits through tar application, formed the basis of our present knowledge of chemical carcinogens. The unit of the vast number of polycyclic hydro-carbon compounds is benzine, structurally a hexagonal arrangement of carbon atoms, each attached to two others and to one hydrogen atom (Fig. 3). Naphthalene ($C_{10}H_8$) consists of a bonded pair of benzine rings. The next substance in order of increasing complexity is anthracene—three benzine rings in a row (Fig. 10). To simplify the nomenclature, the position of the carbon atoms is conventionally numbered as shown. If, to anthracene, two benzine rings are added in the 1:2 and 5:6 positions, we have a substance known as 1,2,5,6-dibenzanthracene (Fig. 11), which was shown to possess to a fairly high degree the ability to produce cancer. Cook and Hieger and their co-workers[11] found another related substance of decidedly greater cancer-producing power and occurring in the cancer-producing fractions of coal tar. It is known as 3 : 4 benzpyrene (Fig. 12). In 1934, methylcholanthrene (Fig. 13) was synthesized and this proved to be even more powerful as a cancer producer than 3:4 benzpyrene. This compound is of great interest from the theoretical point of view: It consists of the 1:2 benzanthracene nucleus to which is added a methyl radical at position 6 and a cyclopentane group at positions 5 and 10. It bears a striking structural similarity to the two principal acids present in bile, cholic acid, and desoxycholic acid; in fact the synthesis of methylcholanthrene was accomplished by four simple transformations from desoxycholic acid.

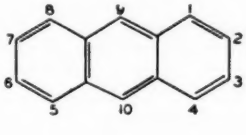
Now, it happens that a number of substances known as sterols, which are of great importance in the body and which are produced physiologically by the body, have chemical structures which suggest the diagrams previously discussed, for example, a female sex hormone from the ovary, estradiol (Fig. 14) and the male hormone, androsterone (Fig. 15). Another hormone from the corpus luteum, progesterone (Fig. 16), has a similar configuration.

These facts suggest that cancer may arise in human beings as a result of reactions taking place within the body whereby normally produced substances, such as the bile acids or the steroid hormones, are converted into cancer-producing compounds such as methylcholanthrene. Of course this is pure hypothesis, as such transformation has not yet been demonstrated. On the other hand, we will see that a relation of sorts seems to exist between estrogenic and androgenic hormones and cancer of certain accessory sexual organs. Further, benzpyrene will produce estrus in castrated female mice, as will another carcinogenic hydrocarbon.



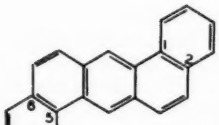
Benzene

Fig. 9



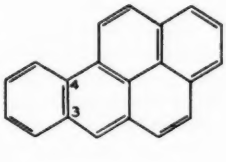
Anthracene

Fig. 10



Dibenzanthracene

Fig. 11

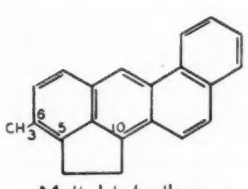


Benzpyrene

Fig. 12

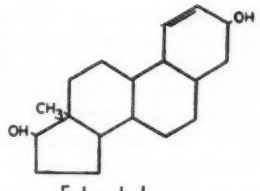
HORMONES.

In 1901, Beatson[12], noting the comparatively more rapid course of breast cancer in younger women, suggested that ovarian function might be linked to breast cancer, and castrated younger breast cancer patients; he thought that in a respectable proportion of patients, the course of the disease was altered favourably.



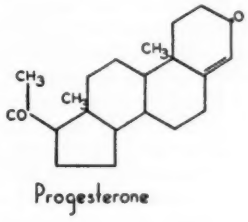
Methylcholanthrene

Fig. 13



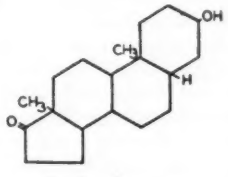
Estradiol

Fig. 14



Progesterone

Fig. 15



Androsterone

Fig. 16

Later, Lathrop and Loeb[13] showed that ovariectomy reduced the incidence of breast cancer in susceptible mice, and that the earlier the operation, the lower the incidence would be. About twenty years ago, Lacassagne[14] discovered that mammary tumors could be made to appear in female mice precociously and, indeed, could be produced in male mice by the injection of estrogenic hormones. A typical growth of the uterine mucosa and fibroid tumors of the uterus and other organs were produced in guinea pigs by injecting estrogenic substances. The list of tumors, attributed by one investigator or another to the exhibition of estrogenic compounds, is now impressive.

The concept that the testicle holds the same relation to prostatic tumors as the ovary holds for breast tumors was supported by a number of statements citing the extreme rarity of cancer of the prostate among castrated patients. The principle demonstrated by Huggins and Johnson[15] in treating advanced prostatic cancer by castration and/or the administration of stilbestrol is now an accepted part of urologic practice. In a high proportion of men with inoperable disease, such management will effect varying degrees of growth inhibition, including decrease in the size of the gland, relief of pain and obstruction, lowering of the serum acid phosphatase, diminution in the size of metastases, and often recalcification of osteolytic bone lesions. In time, resistance to therapy usually supervenes.

Castration and estrogen administration have proved rather consistently useful in providing symptomatic relief and in prolonging lives of men with cancer of the breast.

The animal experimental work in mammary carcinoma, as well as the occasional favour-

able alteration of the course of human breast cancer following castration, suggests the feasibility of chemical (hormonal) ovarian inhibition or castration by means of androgen administration. The evidence accumulated over the past ten years is that male sex hormone in the form of testosterone propionate will excite varying degrees of therapeutic response in about two-thirds of women with advanced and disseminated mammary cancer. In about one-third of those with skeletal metastases, bone repair can be demonstrated. Occasionally, dramatic regression of pulmonary metastases occurs. However, skin metastases and soft tissue deposits in general are usually refractory. Prudente[16] has employed testosterone prophylactically in a series of surgically treated breast cancer patients. In his hands, radical mastectomy alone resulted in a survival rate of 44.4 per cent. in patients judged operable, that is, in clinical stages I and II. Employing testosterone following radical mastectomy increased the five-year survival rate among a group of 79 patients in the same stages to 67.1 per cent. In a group of eleven patients in Stages I and II treated by radical mastectomy and testosterone, the ten-year survival rate was 54.5 per cent. as compared to 18.1 per cent. among those treated only with surgery. Unfortunately, this report makes no attempt to correlate dosage with the results. It is said, however, that testosterone propionate was given in weekly doses of 25 to 350 mg. and extended from five to fifty-eight months, with total amounts ranging from 350 to 16,000 mg.

In 1947, Nathanson[17] upset what looked like an orderly hypothesis by calling attention to the beneficial response of some breast cancer patients to estrogens. While this female sex hormone appears to be generally less useful than the male in the therapeutics of breast cancer, the fact is that it is perhaps more consistently effective against soft tissue lesions and tends to superiority in older women. However, the pragmatic choice based on the patient's relation to menopause is not wholly satisfactory, although it is currently widely employed.

At the moment, cancer seems to be one of the few afflictions of man not substantially benefited by the adrenocorticotrophic hormone of the hypophysis. The biological effects of ACTH and of compounds E and F among the adrenal corticoids which it stimulates are, in fact, profound and broad-ranged. At the moment, cancer does not share significantly in the clinical dividends of ACTH and cortisone. However, these substances have provided a useful method of treating acute leukemia. They will induce remissions of varying length in 50 per cent. to 60 per cent. of children with acute leukemia, and the peripheral blood picture may appear normal in such cases.

Recently, Hertz[18] of the National Cancer Institute, has demonstrated a growth-inhibiting effect of progesterone on epidermoid cancer of the cervix. While the results have been quite inconsistent in the small series so treated, this extension of organotherapy appears to be highly significant on theoretic grounds alone.

Chemotherapy.

Renewed efforts to find chemotherapeutic agents effective against cancer followed the accidental discovery during the war of the preferentially toxic effect of a nitrogen mustard compound on hematopoietic tissues. Attempts to identify, among thousands of possible cytotoxic compounds, those with effective, specific, growth-restraining properties are being vigorously pursued. A scholarly statement of the problem has been presented by Haddow[19]:

> . . . it has always been a matter for legitimate doubt whether a therapeutic agent could impair the growth of malignant cells without equally damaging the normal cells and especially those which are engaged in active division, for example, in the intestinal mucosa, the bone marrow, and the generative organs. There can be no misunderstanding as to the almost insuperable problem which the chemotherapy of cancer presents, and which, in search of a comparison, we can liken to a biological counterpart of squaring the circle.

He adds:

> It is indeed true that those who have considered the matter most thoroughly are under the least illusion as to its practicability.

Nitrogen mustards have been described as radiomimetic, since their toxological effects in normal animals resemble in almost all respects those of total body irradiation. The most extensively used compound in this group is methyl-*bis*-(B-chloroethyl) amine hydrochloride. Its greatest usefulness appears to be in the treatment of Hodgkin's disease as an ancillary to orthodox irradiation management. It will induce subjective improvement in the majority of patients with Hodgkin's disease and objective response in many of them. The average duration of remissions is six to seven weeks. Some patients have received ten and more courses of treatment with repeated clinical relief, but, in all, the disease sooner or later becomes refractory to treatment, and there is little evidence that life is appreciably prolonged. It has been found to produce transient improvement in about half of patients with inoperable cancer of the lung, and occasionally shrinking of tumors, absorption of effusions, and aeration of atelectatic areas may occur. In summary, nitrogen mustard is an effective, temporary,

palliative agent of irregular activity in Hodgkin's disease, lymphosarcoma, chronic leukemia, polycythemia vera, mycosis fungoides, primary lung cancer, and, to a much lesser degree, in other miscellaneous neoplastic disorders.

A recent development, triethylene melamine, shows early promise of therapeutic effectiveness approaching or equalling that of HN_2, with the advantages of oral administration and absence of nauseating effect.

In 1944, it was reported that injections of a crude extract, which had many of the properties of folic acid, would inhibit in mice the growth of transplanted sarcoma, and subsequently, that the same activity was shown by pteroyltriglutamic acid, a conjugate of folic acid. Within three years, a number of reports appeared attributing palliative usefulness to conjugates of folic acid, notably Teropterin, including increase in energy, appetite, sense of well-being, and, indeed, pain relief.

Previously, Spies and associates[20] had shown the important role of folic acid in hematopoiesis, which was in accord with the fact that in mammals the induction of folic acid deficiency by withholding that compound from the diet results in anemia and leukopenia. The principle of antimetabolite effect suggested an interesting possibility for the induction of a folic acid deficiency in tumor-bearing individuals through the administration of compounds so similar to folic acid that the cells would take them up as folic acid, but at the same time so dissimilar that they could not function as folic acid in cytochemical activities. Antagonists were rapidly found, and, of them, 4-aminopteroyl glutamic acid (Aminopterin) has probably been most extensively employed. Early its usefulness was found to be greatest in acute and subacute leukemia, of which cases from 40 per cent. to 60 per cent. show varying degrees of remission, always temporary. It is one of the paradoxes of medical progress that at a symposium held in 1948 at the New York Academy of Medicine both folic acid and its antagonists were advocated simultaneously as treatments for cancer.

The list of useful chemotherapeutic agents is not long. Urethane, a drug of low toxicity, used in the past as a hypnotic, produces remissions in chronic myelogenous leukemia, similar to those induced by x-rays, radiophosphorus, and nitrogen mustard. Its effects on chronic lymphatic leukemia are less predictable. In general, its action is less precise and less consistent than that of the more toxic agents and it is unlikely to be effective when the latter fail.

Stilbamidine and a derivative, pentamidine, are new drugs which have proved effective in the treatment of kala-azar. Because patients with kala-azar have a high serum globulin, Snapper[21] tested the effects of stilbamidine in multiple myeloma, also characterized by high serum globulin levels. The results of these drugs are limited but definite, and the most important is the relief of bone pain in the majority of cases, often of extended duration.

It is unnecessary to discuss here the recent advances of radiation therapy and surgery pertinent to cancer treatment, and I shall not extend my remarks on these subjects beyond the following: Experience with radioactive isotopes does not appear to have justified earlier enthusiasm for their potential role in cancer therapy. Radiophosphorus has a limited place in the management of a few neoplasms of the hematopoietic system; radiosodium may be the treatment of choice in polycythemia vera; radioiodine is of definite value in occasional types of thyroid carcinoma, but less than 20 per cent. of such tumors show the necessary affinity for iodine (indeed, radioiodine appears to have greater usefulness in diagnosis than it has in treatment); radioactive gold, by virtue of its inertness, is being used experimentally, in local tumor deposits; radioactive strontium and calcium have been tried without success in tumors of bone. Radioactive cobalt appears to possess essentially the same radiobiologic effects as radium and this, together with its greater availability and low cost, appears to make it a valuable substitute for radium.

The advantage of x-rays generated at 1,000 and 2,000 kilovolts is established and a slowly increasing number of supervoltage machines is recorded. Their capacity for delivering relatively larger proportionate depth quantities of energy combined with greater targeting precision have increased the range of effectiveness of x-rays, but the greater penetrability of such rays offers new hazards of carcinogenicity for deep tissues.

Abetted by antibiotics, better anaesthesia, and improved control of fluid, electrolyte, and nutritional dislocations, surgery continues to seek new cancers to conquer. A clear example of the evolution process is seen in the steady decline in the operative mortality in gastric resection. Of the first 37 patients subjected to resection, 73 per cent. died operative deaths. Today, in expert hands, the mortality of total resection is less than that of the limited procedures of twenty years ago. So safe has it become, a leading surgeon has recently suggested that total resection be the rule in treating gastric cancer.

Examples of the heroic procedures being currently employed are resection of the mandible and floor of the mouth combined with *en bloc* radical dissection of the neck; applica-

tion of previously recognized surgical principles based on lymph drainage pathways to more radical efforts to circumscribe melanoma and pelvic exenteration operations for advanced cancer of the rectum, prostate, bladder, and uterus. Brunschwig and Pierce[22] have recently reported the early returns based on the first hundred exenteration operations performed for inoperable cancers of the cervix, corpus, vagina, and rectum procedures involving, in the majority of cases, total hysterectomy with pelvic lymph node dissection, cystectomy, proctosigmoidectomy and ureterocolic anastomosis. Operative mortality, defined as death within one month regardless of cause, was 20 per cent. Forty-nine lived beyond one month, but not beyond eight months, and 35 of these died of metastases not apparent at the time of operation. Nine of the hundred obtained effective palliation and survived at least one year, but eventually succumbed to metastatic disease. Twelve have survived at least one year and five months, have returned to normal activity and showed no disease at the time of reporting.

Wangensteen[23, 24] is engaged in an interesting effort to increase survivorship among patients who have previously had colonic resections. Resected patients are explored at the end of a given postoperative interval, usually four to six months, even in the absence of signs of recurrence and symptoms. This he calls the "second look." In a few cases resectable recurrent disease has been present.

In this rambling and incomplete survey of what we may call the frontier of applied cancer knowledge, I hope enough has been presented to illustrate, first, the enormity and the complexity of the problem which, in fact, appears to be expanding exponentially with our increasing knowledge; and, second, the contemporary quickening of interest in the problem among all of the major scientific disciplines.

If I may venture some philosophical remarks, I would point out the extreme breadth of the spectrum of research effort being applied directly and indirectly to the problem of abnormal growth. It ranges from investigations of the most fundamental sort, having apparently little to do with growth, to a direct assault on human cancer. These two extremes, often referred to as "basic" and "applied" for science in general, find felicitous expression as "research and development" in the name of an important office concerned with modern military technology. A frequently discussed question is whether either is the path of choice in seeking the earliest possible control of cancer. The advocates of the applied or clinical approach hold that effective therapeutics have been discovered for a number of diseases well in advance of our knowledge of the essential nature of these diseases —digitalis and quinine are the stock examples. The fundamentalists, on the other hand, call such tactics "wildcatting," and while admitting that a lucky shot in the dark might hit the mark, point out that much of what we know about cancer has been derived from basic studies in areas which appeared unrelated. Moreover, it now seems unlikely that the genesis of cancer will be unitary, but, rather, will prove to be the resultant of several or numerous vectors. It is also highly probable that a single effective treatment for the group of diseases called cancer is as improbable as a universally effective antibiotic. This disagreement as to the "best" method of attack is one example of the many hazards which beset the investigator.

There are many thoughtful observers of the field of the biological sciences who, while conceding that the ultimate solutions of the vast problem of abnormal growth will come to hand, are, nevertheless, of the opinion that it will not happen suddenly nor soon. There is reason to believe that cancer will not be undone by a bolt from the blue, by any dramatic and decisive break-through, but rather will it give way before persistent research attrition. We must resign ourselves to whittling away at this mass of mystery, and gradually, imperceptibly, the truth will emerge, so that an historian of the future, writing the record of mankind against cancer, will not be able to fix the day nor the month nor the year when victory was achieved. Our understanding of cancer's causes, and the development of effective preventive and therapeutic measures may well prove to be a more evolutionary process than we would like, but so has so much of what we know today.

Control.

But while we continue to wait for the accumulation of coherent and utilitarian facts by the forces of research, there is much that we can do to achieve a respectable degree of control over cancer, employing the knowledge and techniques which we already possess. In fact, I consider it doubtful whether any major medical problem facing the people of our country is more susceptible of immediate and substantial relief than cancer. Much progress has been made—most of it within our lifetime. It is not enough to make us complacent, but still enough to show that we are not pursuing a will-o'-the-wisp. May I remind you of the scene forty years ago. Cancer was eighth in the list of causes of death, preceded by heart disease, tuberculosis, pneumonia,

Bright's disease, diarrhoea and enteritis, and diseases of the vascular system. Yet 78,000 people a year were dying from it. What research there was was more of alchemy than science. There was but one specially designated cancer hospital and there were no cancer clinics. There was no support from the federal government to programs of research or control and only one state recognized its responsibility in this respect. No word of cancer appeared in the media of public information. Nowhere was cancer a reportable disease. The biopsy was still a subject of controversy among doctors. X-rays were being employed somewhat gingerly and radium cost five times its present price, thereby limiting its availability greatly. The operation for cancer of the rectum was an innovation and tumors of the central nervous system, of the lung, and of the pancreas were not yet surgical diseases. Anaesthesia was limited to two or three agents of limited flexibility. Medical education was unstandardized and diplomas from "mills" afforded purchasers the same rights and privileges as were open to the graduates of the first-class schools.

Today cancer is second among the causes of death and there are about 220,000 deaths per year from it. The country-wide budget for cancer research in the United States is in the order of $15,000,000 annually. The American College of Surgeons approves 11 cancer hospitals, 539 cancer clinics, and 113 cancer diagnostic clinics. This year's appropriation by the federal government for cancer research and cancer control exceeds $19,000,000, and the voluntary health agency, the American Cancer Society, last year raised $16,500,000 for these purposes. Cancer information for the public is given wide attention in newspapers, magazines, on the radio and television. X-rays are being generated at higher and higher voltages and targeted with increasing precision, while radium and its new substitute, radioactive cobalt, are available generally throughout the country. Surgery has boldly extended its frontiers so that there is virtually no part of the body now inviolate before the scalpel.

Virtually everything we know today about cancer has been learned in a single generation. Yet we are still in the green years of medical progress, and we have no reason whatever to feel resigned nor apathetic in respect of future progress in the control of this disease. I believe the crucial point in our contemporary effort to control cancer is to be found in the dichotomy which exists between the curability of cancer under optimal conditions of localized disease and expert treatment and the cures actually being achieved on the other (Table IV). The difference between cures now possible and those being generally achieved is in important degree due to the delay between the onset of cancer's signs and symptoms and the institution of definitive treatment, which delay remains appreciable in most cases. No other disease presents an even similar dichotomy, and no other disease pursues a more uniformly fatal natural course, and no other disease has more trifling beginnings.

TABLE IV

POTENTIAL VERSUS ACTUAL RATE OF FIVE-YEAR SURVIVALS FOR SELECTED SITES OF MAJOR CANCER

	Localized	Average Allcomers
Breast	80	35
Cervix	70	30
Larynx	90	15
Rectum	75	12
Stomach	60	5
Lung	50?	4-

The early apprehension of these quiet and indistinct beginnings is possible only by the patient himself. Therefore, if we are to reduce the disparity between cures possible and cures accomplished, we must create a pervading awareness of cancer's early signs and of the importance of immediate medical attention. This is the purpose of lay education or cancer propaganda, as its critics like to call it. This is attempted, chiefly by the American Cancer Society, through the media of pamphlets, posters, exhibits, motion pictures, lectures, radio, television, newspapers, and magazines. There are those who charge that cancer propaganda is harmful. They are themselves divided into two camps; those who say that cancer publicity is scaring people away from doctors' offices; and those who state that the same publicity is frightening people into doctors' offices needlessly. Their watchword is cancerophobia.

Of course, cancerophobia is not new, and we have reason to believe it antedated the American Cancer Society. Over two hundred years ago, Vacher reported a curious instance of widespread fear of cancer:

> In the year 1734 suddenly the whole female population of Besancon was overcome with the fear that they were suffering from cancer of the breast or might be so affected in the future. This followed a surgeon's suggestion to the women of that city that they examine their own breasts for lumps. The sequel to this announcement was that all women examined their breasts so often and so long, and squeezed them so much that a certain number really did develop lumps which were subsequently speedily removed with greatest success by the surgeon.

In order to examine the validity of the charge that cancer propaganda is harmful to the general public in producing an unwarranted increase in the level of fear and anxiety, opinion was sought by the American

Cancer Society of a 10 per cent. sample of the Diplomates of the American Board of Psychiatry and Neurology (Table V). The results may be summarized by saying that 89 per cent. of the sampled group did not express disapproval of cancer public education, while 11 per cent. were unequivocal in their disapproval. Apparently the harmful effects of cancer publicity are not so serious as our more vocal critics would have us believe. Many of the respondents pointed out that phobias and neurotic attitudes are not determined by external situations, such as reading a pamphlet, but rather do they arise in subconscious mental mechanisms to which certain individuals are predisposed and which lie infinitely deeper than mere publicity can possibly go. Such persons are bound to find something to fear, and if it is not cancer, it will be insanity, heart disease, the atom bomb, or their spouse's fidelity.

TABLE V

SUMMARY OF OPINIONS TOWARD CANCER PUBLICITY AND EDUCATIONAL MATERIAL ATTITUDE TOWARD CANCER MATERIAL

	Number	Per Cent.
Definitely disapprove as harmful ..	31	11.4
Does some harm, but no opinion on whether good outweighs harm ..	21	7.7
Does some harm, but good outweighs harm	44	16.2
Does no harm (positive good implied in majority of these)	117	43.0
No opinion (including "not qualified to judge")	59	21.7
Total	272	100.0

A serious effort to discover cancer before it gives rise to signs and symptoms has been made in this country during the past decade, particularly during the past four years. The large-scale examination of symptomless persons for evidence of early cancer was begun simultaneously in 1938 by two women physicians, Catherine MacFarlane of Philadelphia and Elise L'Esperance of New York. There are at the moment upward of 240 detection centres in various parts of the country, and, in addition, there has recently arisen formal expression of the practice of cancer detection in the doctor's office. The significance of this effort for cancer control is brought out in figures recently compiled by us based on 52,000 such examinations conducted in ninety scattered centres. The over-all incidence of cancer in this group of presumably healthy people was 0.8 per cent., or, as we prefer to express it, eight per thousand (Table VI). Among persons 60 years old and beyond, the rate of cancers detected in this group of centres was more than four times greater than the rate for the group unselected as to age. If the objective of the program of cancer detection is to find the greatest possible number of cancers with the limited man power available, there may be reason to restrict the formal program to those over 50. The yield among younger persons is perhaps not great enough to justify the time and money required. Again, it may be well to consider emphasis on certain body sites.

TABLE VI

RATE OF CANCER BY AGE AMONG PRESUMABLY WELL PERSONS, BASED ON ANALYSIS OF 52,000 CANCER DETECTION EXAMINATIONS (AMERICAN CANCER SOCIETY, STATISTICAL RESEARCH SECTION, 1950)

American Cancer Society Detection Centre Survey	
Under 30	Incidence rate 1.2 per thousand
30 to 39	Incidence rate 2.4 per thousand
40 to 49	Incidence rate 3.9 per thousand
50 to 59	Incidence rate 12.4 per thousand
60 plus	Incidence rate 34.3 per thousand

DETECTION TECHNIQUES.

Recent studies suggest that cancer of the cervix may exist for years in a "cocoon" or noninvasive stage and present no gross evidence of disease during that time, although the vaginal smear will indicate it. Recent experience with mass chest x-rays and attention to their cancer case-finding potential have shown that cancer of the lung can and does exist as a "silent-shadow," identifiable in the x-ray film well in advance of the classical symptoms of cough, expectoration, chest pain, etc. Early returns indicate that the rate of resectability in asymptomatic lung cancer is twice as great as it is in patients with symptoms. Again, cancer of the rectum can be felt in most cases, seen through the proctoscope in all, when it is no bigger than a pencil's eraser. At this stage of growth there are no symptoms; the classical signs—change in bowel habits, bleeding—will come later. The average breast cancer has reached a diameter of 4.5 cm. before it excites the patient's serious attention, and by this time over half have metastasized; yet it is possible for any intelligent woman, as well as any intelligent doctor, to find a lump a quarter that size provided the trouble is taken to feel for it. The listing of types of cancer which can today be found early enough to justify vastly increased hopes of cure would properly include those of the oral cavity and of the skin, since both sites are foremost examples of accessibility and simplicity of instrumentation. Now, the fact is that these six sites of cancer origin—cervix, lung, rectum, breast, mouth, and skin—account

for nearly 50 per cent. of cancer deaths among women and about 25 per cent. of cancer deaths among men. Cancer originating in these sites presents these features: (1) each is now susceptible of discovery while it is small and asymptomatic; (2) each has a high rate of curability when properly treated while small and asymptomatic. Given the conditions of a small, asymptomatic, localized lesion and adequate treatment, cancer of the cervix could be cured four times as often as is the case generally at present; cancer of the lung and of the rectum, five times as often, and cancer of the breast, twice as often.

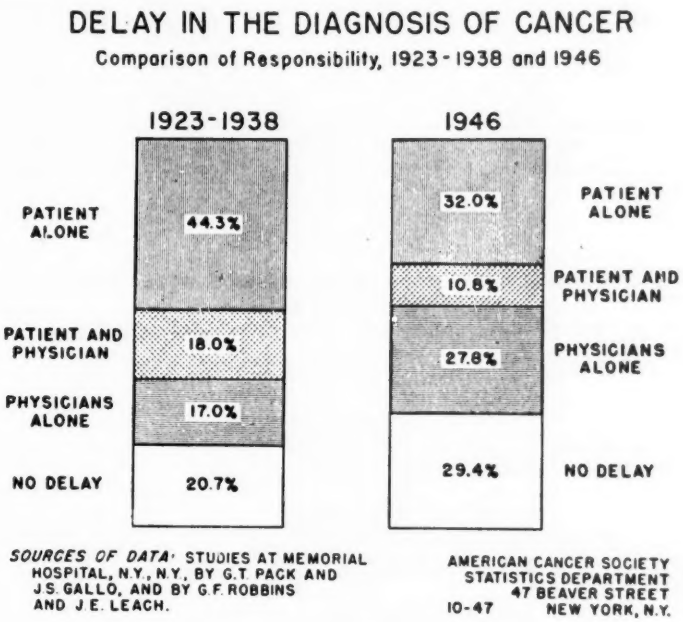


Fig. 17

TABLE VII

PERCENTAGE OF BREAST CANCER PATIENTS WITH AXILLARY METASTASES
MAYO CLINIC, ROCHESTER, MINN.

Year	Per Cent. with Axillary Disease
1910-1919	63.6
1943	47.0

(From Harrington: J. Michigan State M.Soc., January, 1948.)

RESULTS.

Scattered returns from a number of sources suggest that the program of cancer control which I have laid out in some detail is no wild gleam in a health educator's eye, but is a valid objective worthy of the support and participation of all. First, it appears that the important and often critical factor of patient delay is being influenced in the right direction (Figs. 17 and 18). This reduction in patient delay appears to be reflected in the experience of the Mayo Clinic, where, in general, the proportion of patients with breast cancer in Stage I at the time of treatment appears to be increasing (Table VII). Our own study in Vermont (Table VIII) showed that the number of patients with apparently localized disease had nearly tripled in a ten-year period. Dublin's analysis of the cancer death rate among female policy-holders of the Metropolitan Life Insurance Company showed that there had been an 11 per cent. decline in the over-all cancer death rate in the decade following 1936 to 1938, and in the group between 55 and 64 years of age the decline amounted to 15 per cent. (Fig. 19). Data from

TABLE VIII

PERCENTAGE OF HOSPITALIZED CANCER PATIENTS WITH LOCALIZED DISEASE
VERMONT — 1937 AND 1947

Year	Per Cent. with Localized Disease
1937	20
1947	58

In 51 per cent. of patients with cancer admitted to Vermont Hospitals in 1947, therapy was undertaken with curative intent. (From: American Cancer Society, Vermont Cancer Study, 1948.)

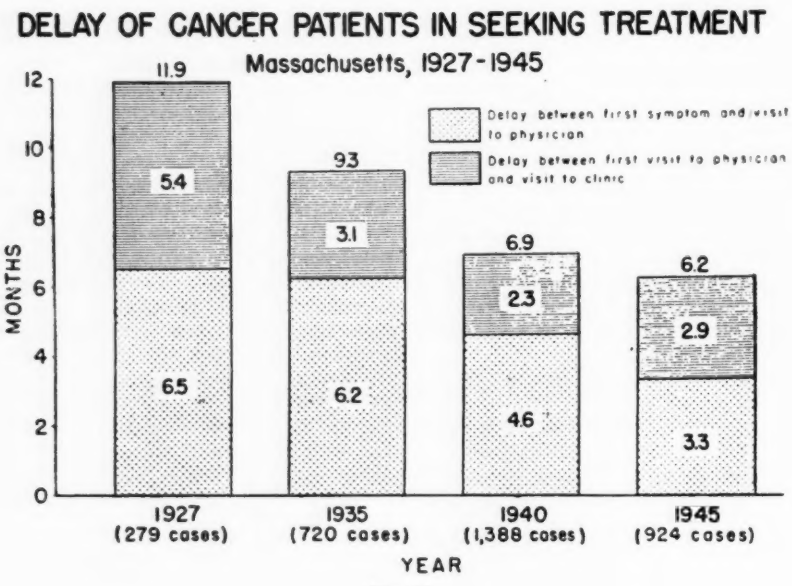


Fig. 18

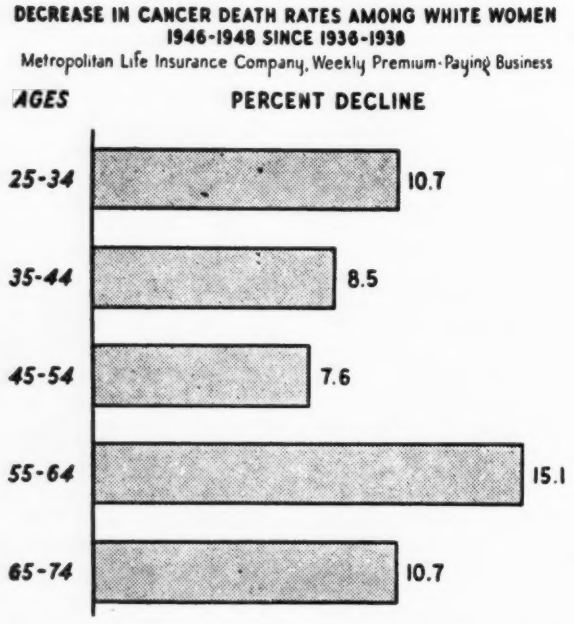


Fig. 19—From Statistical Bulletin, Metropolitan Life Insurance Company, vol. 30, no. 3, March, 1949.

the extensive and highly accurate Tumor Registry in the State of Connecticut disclose consistently rising survivorship rates among both male and female cancer patients (Figs. 20 and 21).

I have tried in this somewhat rambling and, I fear, overlong survey of the cancer scene to arrive at a point of view with perspective and of long range. I have presented the prospects and the difficulties in research, but I have also pointed to the potentials of our modern therapeutic technology. I have, finally, to offer for your continued reflections, this conviction: A respectable measure of control over cancer need not wait for dividends of future

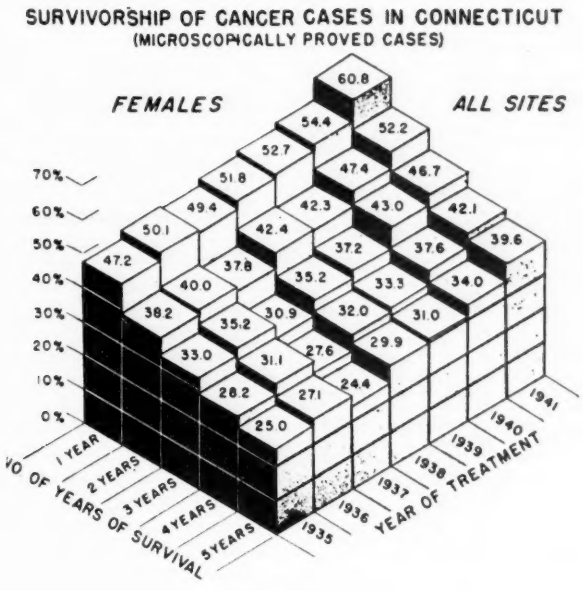


Fig. 20

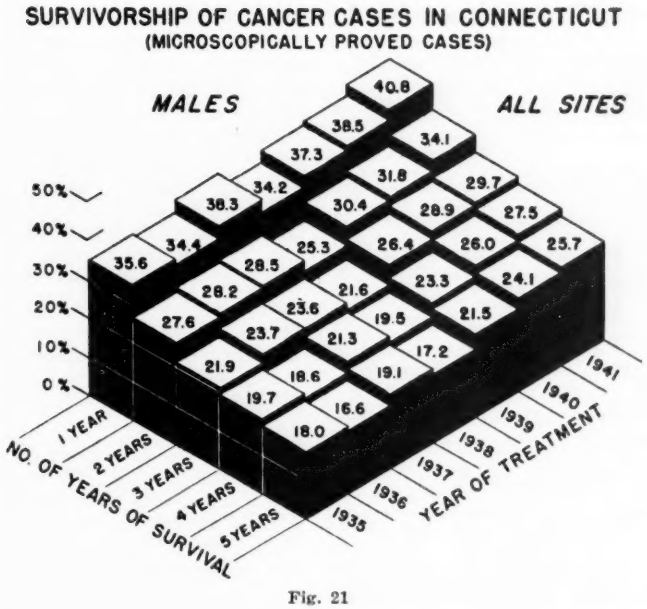


Fig. 21

research nor for extensions of our technical abilities; it waits for one thing above all—a general aceptance of the virtue of widespread social responsibility toward efforts of control. We cannot do with it what we have done with the previous leading causes of death. We cannot legislate against it; we cannot consign it to sanitation experts; we cannot rely on wonder drugs. We have to accept it as a social concern, for, insofar as its control is possible by social action, cancer is a social disease. And who is better fitted to lead the way to this perspective than the doctor himself?

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Dental Materials

*Current Notes (No. 20)**

Local Anaesthetic Solutions.

The quality of some local anaesthetic solutions available on the Australian market has been the subject of a number of complaints. The result of this has been two-fold and exemplifies one way in which Australiian dental standards are brought about.

Firstly, a systematic examination of local anaesthetic solutions as obtainable on the "open market" has been undertaken at the Bureau of Dental Standards. Secondly, with the support of the Australian Dental Association, it has recently been agreed to appoint a Sub-Committee of the Standards Association of Australia to deal with dental biological products, and standards for local anaesthetic solutions will be one of its first objectives. At a later stage, no doubt, the Dental Association will consider such products for accreditation as it has already done for alloys, cements, plasters and other such materials. The practitioner in buying a certified anaesthetic solution will then be assured that it is subject to proper control throughout its preparation and packaging and that it is true to label.

So far, the methods of test used at the Bureau of Dental Standards in the testing of anaesthetic solutions have been based on those set out in various pharmacopoeias and those used by the American Dental Association Bureau of Chemistry. The tests include an estimation of anaesthetic agent, vasoconstrictor, pH value, titratable acidity, total solids, residue on ignition, and sterility. Other tests are applied according to whether the material is supplied in ampoules, cartridges or multiple dose containers.

The tests carried out involve those factors which might impair the efficiency and safety of the solutions as customarily used by the dentist. Apart from this the Bureau of Dental Standards is not in a position to comment on the relative merits of various anaesthetics and vasoconstrictors. The questions of choice of anaesthetic agent, vasoconstrictor and dosage and of toxicity are matters for clinicians and pharmacologists to advise upon. The chief concern of the Bureau is, first, to determine whether the actual composition of the solution agrees with the formula indicated by the

*Contribution from the Commonwealth Bureau of Dental Standards.

manufacturer or distributor and, second, to check whether the condition in which the product is supplied is acceptable. If a dentist uses in accordance with recognised practice a material which meets these requirements he should be confident that it will have the desired effect. If it does not then he must first look for other reasons.

In case it may be thought that this is merely an academic matter it should be mentioned that local anaesthetic solutions tested have exhibited such extremes as over twice the stated content of cocaine in one case and virtually no anaesthetic agent in another. These cases are not at all representative but the very fact that such accidents, as they obviously are, can occur and not be detected before distribution stresses the need for appropriate standards and adequate control testing of batches before release to the dental depots and the profession. Leaving aside such gross lapses, which at present are unlikely to be detected until a dentist lodges a complaint, it remains to be seen just how far current supplies of anaesthetic solutions in Australia measure up to the A.D.A. and other specifications.

*Current Notes (No. 21)**

A Statement on Self-Hardening Resins.

The subject of one of the papers presented at the Australian Dental Congress in Brisbane was "The Present Status of Self-hardening Resins in Conservative Dentistry." The author, A. R. Docking, wishes to emphasise through the medium of "Current Notes" some points which were not, apparently, fully appreciated by some of those who attended.

A full account of the lecture will be published in some other form but, in order to make this statement intelligible to the readers who were not present, an outline of the lecture will first be given.

As the virtues of self-hardening restorative resins are so well known it was considered advisable to use the time allotted in an analysis of the reasons for the reported waning popularity of the resins in the U.S.A. and Continental Europe and in determining the relevance of this to Australian practice. In view of these disturbing overseas reports a general note of caution was sounded and a plea made for careful clinical work and observations. It is this attitude, and not one of out-of-hand rejection, that is necessary here in fairness to some of the more promising developments in techniques and materials for which there has not been adequate time for conclusive clinical tests. For example, results suggest that acrylic fillings placed in cavities properly lined with zinc phosphate cement are not nearly so frequently troublesome as those placed without a lining.

The criticism of self-hardening restorative resins was based on the results reported from extensive clinical surveys and from discussions with dentists here. The difficulties resolved into two which have been found far more common than any others; these are discoloration and poor cavity seal. The former and more common complaint is now thought to have been successfully mastered by the use of catalysts of the toluene sulphinic acid types. There remains the question of leakage around the margins which manifests itself more insidiously as a softening of the cavo-surfaces with associated odour and taste. It was with this problem that most of the lecture was concerned.

After giving an account of the phenomenon and the growing consciousness of its seriousness apparent from correspondence and published surveys, the possible causes were examined. As amalgam and silicate restorations do not usually behave in this way it was thought instructive to list the most important relevant properties which distinguish acrylic resins from them. These are: (a) dimensional changes on setting, (b) elastic modulus, (c) coefficient of thermal expansion, and (d) bacteriostatic properties.

(a) Acrylic resins must shrink on setting (polymerising) and if the margins are to be well adapted special precautions must be taken, for example the use of pressure matrices or the brush technique or cavity adhesives. While the proper use of these decreases the marginal leakage at the time of placement the advantage does not appear to be lasting.

(b) The low modulus of elasticity of acrylic resins means that it is too flexible a base for poorly supported enamel rods at the cavo-surface margins. This can be responsible for crumbling and staining at these margins and may contribute to leakage around the entire filling.

(c) Thermal expansion of acrylic resin has come well into the picture recently, chiefly due to the simple demonstration of droplets of fluid forming at the margins of restorations in teeth which have been placed in iced water and later warmed by holding in the fingers. Other tests suggest that this percolation, which is also found with other restorative materials when freshly placed, is not due simply to thermal expansion. While the high thermal expan-

*Contribution from the Commonwealth Bureau of Dental Standards.

sion of acrylic resin is a definite disability there are other factors possibly of greater importance to be considered.

(d) The inertness of acrylic resin means that in service it has no extracted or breakdown substances that can exert bacteriostatic and cariostatic effects. It is in this connection that the use of a suitable lining may prove to be of prime importance for extending the life of acrylic restorations.

Another important factor, particularly for resins accelerated with toluene sulphinic acid, is the maintenance of a dry field during placement and polymerisation. It was also stated that use of the resins should be confined to certain types of restorations which theory and experience indicate are more favourable.

The lecture was concluded with the recommendation to continue to use the self-hardening resins with discretion and to observe closely their behaviour in the mouth. This conclusion still holds but in this statement it is desired to emphasise that, if the certain precautions are taken, there is no evidence yet to show that the acrylic restoration will not give satisfactory service.

Until a synthetic resin is perfected for application in restorative dentistry the best guide that can be suggested is:

1. Avoid cavity types that are contra-indicated.
2. Use products based on the most recently developed accelerators, for colour retention particularly.
3. Follow the manufacturer's directions carefully.
4. Maintain a dry field, using rubber dam if necessary.
5. Line the cavity with a suitable cement.

Case Report

An Unusual Case in Hypnodontics

Master ———, aged 13 years, was brought to me for a simple restoration in an upper left lateral. As two Sydney practitioners had been unable to handle the lad, his mother arranged with me to have the work done under hypnosis.

The patient entered my rooms in a very overwrought and tearful condition and immediately informed me that he didn't want to go to sleep and wouldn't go to sleep. As it is almost impossible to hypnotise a person against his will, I abandoned all thought of hypnosis. After much cajolery, I persuaded the patient to sit in the chair and began to break down the overhanging enamel with a chisel. After a few moments the patient said, "You're making me tired. I'm going to sleep," then his eyes closed and he went into a light trance! Still keeping the chisel on the tooth, I deepened the trance by the usual method and was able to complete the restoration without trouble.

A perfect example of the fact that the operator never hypnotises his subject; the subject hypnotises himself at the operator's suggestion. In this case the patient was convinced that he would be hypnotised and so when I started to do something which he didn't fully understand, he assumed that it was intended to put him to sleep. His keen imagination did the rest.

G. H. Estabrooks records a similar case in his book *"Hypnotism."* In that case the subject went into a light trance whilst listening to a recording of a Swiss yodelling song, simply because he had been previously convinced that the record would hypnotise him.

J. F. Morris, Bombala.

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Editorials

Antibiotics and Oral Infections

The widespread use of antibiotics in medicine and the increasing number of these agents which are currently available provide a means of treatment for oral and dental infections of virtually unknown limits and also as a result of their systemic implications widen the horizon of dental practice. As a necessary consequence of this, the use of antibiotics demands, on the part of the dentist, a wider appreciation of their general effects.

At the outset, it must be stated that antibiotics must not be regarded as a substitute for well established therapy, in spite of the extravagant claims made for each successive agent as it appears. The most commonly used of these chemotherapeutic substances are the sulphonamides, penicillin, streptomycin, aureomycin, chloromycetin (chloramphenicol) and terramycin.

Periodontia, endodontia, dental caries and oral surgery have all been considered within the range of usefulness of these substances. The method of administration of antibiotics varies. In many cases, topical application is suggested; however, the intra-muscular route is advocated either due to the concentration of the agent required or the rate of absorption.

However, there are three possible side effects of antibiotic treatment which must be considered, (a) localised reactions, (b) sensitization to the particular antibiotic, (c) production of resistant strains of organisms.

It is considered that 8-10% of persons are allergic to penicillin and the increase in antibiotic resistant organisms, particularly staphylococci and non haemolytic streptococci, is a well known phenomenon. Some of the other antibiotics have equally harmful effects, the neurotoxicity of streptomycin and the bone marrow aplasia of chloromycetin.

The most common and widespread abuse is the use of antibiotics without specific indications. Not only is such a procedure without value but the toxic side effects of the drugs and the opportunity for resistant strains of organisms to develop make it a hazardous procedure for the patient. Due to the number of antibiotics effective against oral infections, a choice of agents exists. However, on account of the specificity of different antibiotics, the selection of the drug should be made on the basis of culture sensitivity tests.

In the current literature, conflicting reports concerning the effectiveness of different antibiotics appear. For example, penicillin dentrifices have failed in three out of four experiments to have any significant effect on dental caries, yet in one case, achieved, over a two year period, a 50% reduction in new dental caries.

Penicillin is useless for the treatment of herpetic lesions, whereas chloromycetin and terramycin have some effect. Penicillin topically applied will control the organism associated with Vincents Infection, but as Ostrander[1] has observed "This response does not constitute cure and the usual procedures for restoring good mouth hygiene must be carried out".

The local use of penicillin or the sulphonamides in sockets after extraction would not appear indicated.[2]

Polyantibiotic treatment of infected root canals has achieved, apparently, remarkable results in endodontia, but it is suggested that the results are not better than those gained by commonly used drugs.

On the other hand, it is well recognised that antibiotics are valuable prophylactically prior to the extraction of teeth, for the treatment of patients with a history of rheumatic fever, and also for the treatment of osteomyelitis and cellulitis.

The problem of the indications for antibiotics has been well stated:—

> Before administering antibiotic treatment, the practitioner must answer the following questions: Is the infection sufficiently serious to warrant the use of a drug the efficacy of which for critical situations may be impaired by previous administration to the patient? Can the infection be adequately treated by other available means? Have other appropriate measures been taken? Does the specific nature of the infection indicate that the antibiotic will be effective in eliminating the causative organisms? Does the patient's previous history imply that he will suffer no serious reactions from administration of the antibiotic? Does the method of administration appropriate to the specific situation promise a safe and effective outcome? In many cases, if the dentist should ask himself these questions, his answers would preclude the use of an antibiotic.[3]

The whole field of antibiotic therapy is constantly changing and those who wish to use these drugs have an obligation to keep informed of recent developments in order to render the best service to the patient.

It is a field in which the co-operation of the physician and the dentist can be one of great advantage, not only mutually, but in regard to the patient's health and well being.

The choice of the antibiotic and the indications for its use should be considered critically in every case, and the operator should be keenly conscious of the potentialities of the drug to be administered.

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Dental Dissemination of Infective Hepatitis

An increasing awareness of the frequency and the severity of liver damage caused by infective hepatitis has been shown by the recent publication of the Transactions of the Eleventh Conference on Liver Injury.[1]

It has been observed that outbreaks of hepatitis occur after the injections of large groups of persons with blood plasma, serum or some vaccine, a relatively high percentage of them developing the disease.

Of the two types of hepatitis, the chronic epidemic and the serum hepatitis, the mode of transmission of the virus of the latter type gives it a special interest to dentists.

The presence in the community of chronic carriers of the serum hepatitis virus, who have had no previous symptoms, presents a serious problem in relation to transfusion. Professor H. K. Ward[2] has recently pointed out that one in five of the soldiers in Korea receiving blood transfusions are contracting infective hepatitis as a result of blood donors who are virus carriers.

As the virus can only be transmitted by direct skin puncture, the further possibility arises that persons may be infected from the carriers by the use of syringes, needles and scalpels not sterilised by *heat* in between patients.

At Johns Hopkins Hospital, it has been observed that one in every seven patients affected by hepatitis had received a transfusion shortly (5 weeks-5 months) before.

The only effective way to sterilize needles, syringes and scalpels is by heat, and if the dentist relies on cold disinfecting agents for sterilization of these instruments then he runs the risk of transmitting the virus of serum hepatitis from an unknown carrier to some otherwise healthy but unfortunate patient.

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News and Notes

Dental Research

The Dental Board of New South Wales has made a grant of £1,200 to the Institute of Dental Research, United Dental Hospital of Sydney, towards the salary of a research worker, who will be known as the Dental Board Research Fellow.

It is proposed that the Fellow when appointed shall investigate problems of dental decay, a disease which is among the most common affecting civilised man.

Some Facts About the Federation Dentaire Internationale (F.D.I.)

What is the Federation Dentaire Internationale (F.D.I.)?

The Federation Dentaire Internationale (FDI), founded in 1900, is the recognized international organization for the dental professions of the various countries of the world. It is a permanent association of national dental societies and their affiliates. It is a true federation in the sense that there is a central government but that each member national dental society retains its independence in domestic affairs. At the present time, forty national dental societies hold membership in the FDI.

What is the Primary Objective of the FDI?

The original objective of the Federation was to sponsor an international dental congress at regular intervals. As the Federation evolved through more than a half century, the original objective became simply a means of implementing the present broad objective: "the attainment by all peoples of the highest possible level of dental health in its relation to the individual as a whole."

What are the Functions of the FDI?

Within the broad assignment of promoting the dental health of the public and the advancement of the dental profession through the world, the Federation has the following specific functions: to promote an international dental congress every five years; to aid in the

development of strong national dental societies in all countries of the world; to encourage closer relations and the exchange of information between the national dental societies; to publish the *International Dental Journal*; to study and report on dental problems of international interest; to co-operate with other international organizations in the fields of science and health; to recognize, by appropriate awards and prizes, contributions to the advancement of dentistry; to stimulate the establishment of documentary centres for the collection of dental literature.

How is the Federation Financed?

The Federation is financed entirely by voluntary contributions from dental societies and individual dentists interested in fostering sound international relations. More specifically, the Federation is supported by (a) annual dues paid by the member national dental societies; (b) voluntary contributions from individual dentists who enrol as "supporting members" of the Federation; (c) any surplus remaining after the promotion of an international dental congress; (d) contributions of time and effort from its officers and members who conduct the programme of the Federation without financial gain.

Under the present By-laws, member national dental societies pay an annual subscription based on the number of members. This subscription ranges from fourteen dollars for the first 100 members to a maximum $4,200 annually for the larger dental societies.

Owing to the vast programme which still remains to be taken in international dental affairs and owing to the fact that many new demands are being made on the Federation for assistance and guidance in many fields, the present support of the Federation is not adequate. Through the medium of "supporting membership," individual dentists may pay an annual subscription and thus contribute substantially to the support of an international programme in dentistry.

What are the Benefits of Membership in The Federation?

Only by membership in the Federation can any national dental society make its contribution to international progress in dentistry. Many international organisations will co-operate only with other international organisations, and thus the Federation presents the only medium for such co-operation in the field of dentistry. The Federation, for example, is the authoritative voice of the dental profession in discussions with the World Health Organisation, the official instrument of the United Nations dedicated to the improvement of the world's health.

"Supporting membership" in the Federation provides the individual dentist with an opportunity to make his contribution to international affairs not only in dentistry but in the broader fields which relate to improved international understanding and good will. In addition, the "supporting member" of the Federation will receive an annual membership card; an annual certificate of membership; news bulletins on the activities of the Federation; admission to the annual sessions of the Federation and a reduced fee for attendance at the quinquennial international dental congresses; the opportunity to subscribe to the *International Dental Journal* at a reduced rate.

How is the Federation Governed?

The general management and control of the Federation are vested in the General Assembly which is made up of delegates chosen by the member national dental societies. In addition there is a Council which is elected by the General Assembly and which conducts the affairs of the Federation between annual sessions.

Where are the Headquarters of the Federation?

While the Federation is legally incorporated in Belgium, its present permanent headquarters are in London. The Secretary-General is the chief executive officer of the Federation and maintains headquarters at 35, Devonshire Place, London, W.1, England.

How is the Programme of the Federation Conducted?

The programme of the Federation is conducted by the permanent staff of the Secretary-General; by the work of commissions, or committees, appointed to study a specific phase of dental affairs; through the reports presented at the annual business session and through the elaborate scientific programme presented at the international dental congresses. At the present time, the Federation has standing commissions in the field of dental education, dental military services, oral hygiene or dental health, dental science and research and public dental services. Other commissions are formed from time to time to survey new problems as they arise.

What is the Future of the Federation?

The future of the Federation can be outlined only in terms of the support that is given it by the national dental societies and individual dentists the world over. The level of dental health nowhere is fully satisfactory and the standards of the dental profession and of dental practice must be constantly improved. More and more the countries of the world are coming to realise that improved health is a

universal problem and can be solved only by universal exchange of knowledge and co-operation. The Federation Dentaire Internationale is the chosen instrument of the world's dental profession to achieve these goals. If dentistry is to have its proper voice in global discussions and solution of the problems relating to improved health, it must have at its disposal an effective agency for international activities. The Federation is organized to meet this need and, for the future, needs only the unstinting moral and financial support of those who have dedicated their lives to the improvement of the world's dental health.

(This is reprinted from a leaflet forwarded from the FDI, and compiled largely from material submitted by Dr. A. E. Rowlett, Honorary President, and Dr. Gerald H. Leatherman, Honorary Secretary-General, of the Federation.)

Dental Services Anzac Day Committee

On Anzac Day, 1953, 67 members of the Dental Services marched under their own banner, and with full recognition by the R.S.L., for the first time since the inception of the March.

The Dental Services, included as a separate identity in Corps Troops, 2nd A.I.F., was led by Brig. H. McD. Finnie, O.B.E., E.D., present Director Dental Services, A.M.F., and Lt.-Col. J. W. Skinner, E.D., Asst. Director Dental Services, Eastern Command.

Seventy members attended the buffet luncheon at the Real Estate Institute's Hall, Martin Place, and voted it an outstanding success.

A Committee, representing all Associations allied to the profession, had been formed under the Chairmanship of Dr. G. S. Cottee, and was responsible for the whole organization of the venture. Some 1,600 circulars were sent out, and though our numbers appear comparatively small against those figures, nevertheless each year adds to the roll-call: 1951—15, 1952—35 and 1953—67. Next year the Committee hopes that those who may have been dubious of the number marching will help swell it to over the hundred.

Excellent radio coverage was given us and the following information made available to the four leading radio stations in Sydney was given word for word, and in two known cases amplified by extra coverage. This not only gave to the profession the news of the Dental Services' support of the March, but to the general public of the part played by the profession in the Services.

Dental Services have played their part in both World Wars and during World War II Navy, Army and Air Force personnel served in every theatre of war.

Army Dental Services were originally part of Royal Australian Army Medical Corps but were separated and formed into Australian Army Dental Corps in 1943. The title "Royal" was bestowed on the Corps for service during World War II.

The Royal Australian Army Dental Corps has the distinction of rendering a more comprehensive service to the front-line soldier than its counterpart in any other army in the world.

It operated in Tobruk throughout the siege and in the fighting in Greece, Middle East, Syria, New Guinea, Solomons and Borneo. It worthily played its part in the Malayan campaign and is now with the Australians in Korea providing the serviceman with treatment equal to that he would expect in civilian life.

The Royal Australian Army Dental Corps is commanded by Brig. H. McD. Finnie, O.B.E., E.D., who leads the March of the Dental Services in Sydney today. He served with the Corps in Greece, Middle East, New Guinea and Borneo.

A photo of our section of the March was arranged and copies may be inspected and purchased at Hall's, Hunter Street, Sydney.

Honour for Sydney Graduate

Dr. Anthony Bull of Sydney has completed a two-year appointment as an Exchange Assistant Professor of Operative Dentistry at Northwestern University Dental School in Chicago. He graduated from the University of Sydney in 1941 and, after serving four years in the R.A.A.F. Dental Corps, he completed the requirements for the D.D.S. degree at Northwestern University in Chicago. He was invited to return as an Exchange Professor in 1951.

At the graduation banquet on June 3, 1953, the Alpha Chapter of Omicron Kappa Upsilon awarded him an honorary key in recognition of his excellent contribution as a teacher during his appointment. The Omicron Kappa Upsilon honour fraternity was founded at Northwestern University in 1914 and now has 42 chapters in dental schools in the United States. The key is awarded for excellence in scholarship and potential leadership to a limited number of the graduating class, and honorary keys are occasionally awarded to members of the faculty or other dentists for outstanding contributions to dentistry.

Dr. Bull returns to Australia with the respect and best wishes of the students and faculty of Northwestern University Dental School, and will take up an appointment as part time teaching fellow at Sydney University.

Charles W. Freeman Resigns as Dean of Northwestern

Charles W. Freeman, Dean of Northwestern University Dental School since 1938, announced his resignation May 11 as Dean; he will continue as Professor of Oral Surgery.

Dr. Freeman has been a member of the School Faculty since his graduation in 1912; he received a Master of Science degree in Oral Surgery in 1925 and became Professor of Oral Surgery in 1933. In 1949 Dr. Freeman was elected a Fellow in Dental Surgery of the Royal College of Surgeons, England.

He is a Past President of the American Association of Dental Schools and the Illinois section of the American College of Dentists.

(Reprinted from Journal of the American Dental Association, Vol. 46, p. 707, June, 1953).

Annual Sports Day

Thursday, 16th July, 1953, was the occasion of the Annual Sports Day of the Association, which was held at the Lakes Golf Club.

Golfers competed for the Sir Harry Moxham Cup and Bowlers for the J. V. Hall Best Bowls Trophy.

A most successful and enjoyable sporting and social day was enjoyed by those members present. Winners in the various events were as follows:

Golf:

Sir Harry Moxham Cup: Mr. J. Newman, square.

Runner-up: Mr. J. Godwin, 1 down.

A. Grade: Mr. A. French, 1 down.

B. Grade: Mr. J. Hardwick, 2 down

C. Grade: Mr. P. Anderson, 5 down.

1st Nine: Mr. D. Ratcliffe.

2nd Nine: Dr. B. J. Smith.

4 Ball Best Ball: Messrs. J. Hardwick and J. Godwin.

Runners-up: Messrs. R. McCrossin and D. Ratcliffe.

Bowls:

Dr. J. V. Hall Best Bowls Trophy: Messrs. J. L. McDermott, W. A. Mitchell, O. Stenmark, H. Hicks.

Runners-up: Messrs. E. Sainsbury, T. Dunphy, J. Moore.

Western Suburbs Dental Group

6TH ANNUAL BALL.

The Western Suburbs Dental Group held their 6th Annual Ball at Petersham Town Hall on Tuesday, 30th June, 1953. It was attended by some 220 members and visitors who enjoyed one of the happiest dental functions ever held.

The President of the Group had a large party of official and personal guests and amongst those entertained by Mr. and Mrs. Frank Dennett were the President of the Australian Dental Association, New South Wales Branch, (Mr. N. E. Edney) and Mrs. Edney, Dr. Frank Helmore and Mrs. Helmore, Dr. Gordon Rowell and Mrs. Rowell, and Mr. and Mrs. Alan Grainger.

15TH ANNUAL MEETING.

The Western Suburbs Dental Group held their 15th Annual Meeting on Tuesday, 21st July, 1953. The meeting was opened by the President, Mr. Frank Dennett, at 8.15 p.m. and the Annual Elections resulted as follows:

Patrons: President of the Dental Board, Dr. J. S. Baird.

President of the Australian Dental Association, New South Wales Branch, Mr. N. E. Edney.

President: Mr. Roy Hawthorne.

Past President: Mr. F. Dennett.

Vice-Presidents: Mr. R. Norton, Dr. R. Cloutier.

Hon. Secretary: Mr. R. Dennett.

Hon. Treasurer: Mr. B. Maundrell.

Committee: Mr. R. G. Leeder, Mr. G. Martin, Mr. W. Mitchell, Mr. H. Sengelman, Mr. D. J. Steele.

Association Activities

Australian Dental Association (New South Wales Branch)

General Meetings.

The Ordinary General Meeting of the Association of 26th May, 1953, was addressed by the first overseas lecturer of this year, in the person of Dr. Roy Rheuben of the United States of America, who delivered a lecture on "Costen's Syndrome." At the same meeting Mr. Brian Piper delivered a lecture on "Long Cone Radiography Technique," a subject which he had studied during a recent visit to the United States.

The second overseas lecturer, in the person of Dr. Earl Pound, addressed the General Meeting of 23rd June, 1953. Dr. Pound had been a guest lecturer at the 13th Australian Dental Congress and, during his visit to this country, represented the American Dental Association. On the occasion of this meeting the Federal President, Dr. J. V. Hall Best, presented to Dr. Pound an illuminated address from the Australian Dental Association. Dr. Pound addressed a large audience on "Functional Aesthetic Dentures."

The Ordinary General Meeting of 28th July, 1953, was the occasion of an address to members by Dr. Earl Bastian, his subject being "Simplified Bridgework." The lecturer used, as part of his illustration, the film recently produced by the Post-Graduate Courses Committee of the Association entitled "Precision Inlays by Hydrocolloid Technique."

On the occasion of these General Meetings the President has taken the opportunity to acquaint members with current Association activities in respect to proposals which had been made to the State Minister for Health to register dental technicians in regard to direct contractual relationships with the public in the construction of dentures.

Executive Report.

Fact-Finding Committee.

The Fact-Finding Committee set up by the Minister for Health to investigate proposals made by dental technicians that they be allowed direct contractual relationships with the public has continued to meet. Meetings subsequent to that reported in the March-April issue of this Journal took place on 22nd June, 7th and 23rd July, 1953.

The representatives of this Association have continued to present to the Committee the views of the profession on these proposals, stressing at all times the danger to the public health if the proposals as placed before the Minister by the dental technicians were implemented.

The indications are that this Committee will continue to meet for some time.

Annual Meeting of the Australian Dental Association.

The Annual Meeting of the Federal Council of the Association was held on Wednesday, 3rd June, in the Senate Room, University of Queensland, Brisbane, during the 13th Australian Dental Congress. This State Branch was represented by Mr. W. A. Grainger (Honorary Treasurer) and Brigadier H. McD. Finnie.

Consideration was given at the meeting to various Annual Reports and a number of matters of general and national import were discussed. The matters dealt with at the meeting will be reported in due course in Federal Newsletters published in this Journal.

Conference with Representatives of Suburban Dental Groups.

On 19th May, 1953, a Conference took place between the Honorary Officers' Committee of the Association and representatives of the Suburban Dental Groups and the St. George Dental Association, at the request of one of these organisations supported by the others.

Discussion at the Conference centred on the establishment of a liaison between the Executive of the Association and the various Groups. Certain decisions of the Conference are to be considered by the various Suburban Dental Organisations and the Executive of the Association.

Post-Graduate Courses.

Three 2-day post-graduate courses were arranged by the Post-Graduate Courses Committee of the Association from 18th to 25th June last. The post-graduate lecturer was Dr. Earl Pound and these courses were most appreciated by those members attending.

Many appreciative comments have been expressed as to the excellence of the courses conducted by Dr. Pound.

Conjoint Meeting of Executive and Delegates from Divisions.

On 6th July, 1953, the Delegates from the various country Divisions of the Association met during the day. Following discussions at this meeting certain recommendations were considered, in conjunction with the Executive, during the evening.

As a result of this meeting a number of constructive suggestions and recommendations will be considered by the Executive at subsequent meetings.

Dental Technicians (State) Award.

Before the proposed conference to consider the terms of the application by this Association for a new Dental Technicians (State) Award could be arranged, the Dental Technicians' Association submitted an application on their own behalf. This application was considered by the Conciliation Committee and a conference will be held to consider both applications. Points in dispute will then be adjudicated by the Dental Technicians (State) Conciliation Committee.

Poisons Advisory Sub-Committee.

The Poisons Advisory Sub-Committee which has been appointed by the Minister to advise as to Regulations and Schedules under the new Poisons Act has been meeting continuously. The dental profession has been represented on this Committee, at the request of the Association, by Dr. E. R. Magnus. Matters debated by the Committee are confidential and it is reported that recommendations of the Committee will shortly be submitted to the Minister.

MEMBERSHIP.

New members.

Calnan, Edward Joseph, B.D.Sc.; Cassidy, Kenneth John, B.D.S., D.D.S.; Colligan, Leslie Arthur, B.D.S.; Couston, John Andrew, B.D.S.; Harley, John, B.D.Sc.; Kidd, Lindsay Gordon, B.D.S.; McGuigan, Maxwell Andrew, B.D.S.; Mitchell, John Francis, B.D.S.; Mumford, George, B.D.S.; O'Connell, William Francis, B.D.S.; Oravski, George, B.D.S.; Scott, John William Albert, B.D.S.

Leave of absence.

Mathews, R.; McEwan, M. A.; McKenzie, G. E.

Student Associates.

Gifford, Allan John; Hallett, Charles Robert; Hoffman, Ronald Arthur.

New Books and Publications

PRINCIPLES AND TECHNIQUE OF EXODONTIA, by F. W. Rounds and C. E. Rounds, St. Louis, 1953, C. V. Mosby Co. (407 pp., 365 illus.). Price £5/5/0. *Our copy by courtesy of W. Ramsay (Surg.) Pty. Ltd.*

A book in which the Principles and Technique of Exodontia are well described is a valuable addition to the library of the dental practitioner.

In this book, the Chapters dealing with the anatomical landmarks and the armamentarium disclose a simple yet adequate approach to the science of exodontia by the authors. However, some of the standard forceps described are too cumbersome for the techniques employed in exodontia in this State.

The section dealing with history, examination and evaluation serves as a useful reminder of the fundamental procedure in exodontia to be adopted by the dental practitioner.

The illustrations in the section dealing with pre-extraction considerations do not well portray the legend. These illustrations, pages 92 and 96, could well be eliminated from the book.

The sections providing a description of the techniques employed for the removal of various teeth are interesting and well described. However, the extraction technique for certain teeth cannot be accepted by all practitioners.

—A. J. Arnott.

THE CLASSIFICATION AND TREATMENT OF INJURIES TO THE TEETH OF CHILDREN, by R. G. Ellis, Chicago, 1952, ed. 3, Year Book Publishers. (247 pp., 164 illus.). *Our copy by courtesy of the publishers.*

A further edition of this book, which deals with a restricted field, is evidence of its popularity and its value, both to the general practitioner and the dental student.

The various traumatic injuries are classified into nine groups, and the discussion and treatment of each group is prefaced by a concise summary of the suggested procedure, which enables quick reference to be made to any section.

There have been minor changes in the text and additions to the bibliography incorporating current trends in techniques and treatment. The use of the pin inlay and pin veneer type of restoration have been dealt with in greater detail, and their many advantages over other types of restoration warrant this emphasis. Developments such as the use of Grossman's polyantibiotic treatment in root canal therapy, and the hydrocolloid techique for construction of gold-acrylic crowns and similar restorations out of the mouth, have been included.

One other desirable addition is the short section discussing ways and means of obliterating the root canal having a wide-open apex. This is always a difficult problem in a young patient, and a logical technique is presented.

The text is well-illustrated with a profusion of clinical photographs, radiographs, and diagrams.

—W. D. Suthers.

DENTAL SURGERY AND PATHOLOGY, by J. F. Colyer and Evelyn Sprawson, London, 1953, ed. 9, Butterworth & Co. (1115 pp., 1015 illus.). Price £5/2/6. *Our copy by courtesy of the publishers.*

The ninth edition of this well known reference text book has been considerably revised and brought up to date.

While one does not always agree with the methods of treatment put forward, and while perhaps this particular work has always attempted to cover too wide a field, it is, nevertheless, an extremely valuable reference book.

The sections dealing with pure pathology are concise, well written, accurate, but it is in the sections on treatment that methods are presented which sometimes do not accord with modern Australian thought.

This edition is very adequately illustrated, and the illustrations are generally of a very high standard. The only discernible weakness in this connection is that the radiograms are presented as "positive," which requires a mental adjustment on the part of the practitioner who is accustomed to diagnose from "negatives."

The chapter on Fractures has been modernised to the extent of including pin fixation, but there is no mention of plating or fixation by Kirschner Wires. It would, however, provide adequate guidance as to treatment for all but the specialist.

The chapter which has been added to the use of antibiotics is also out of date, because of the rapid development in this field, but this weakness is common to all text books, which cannot hope to remain abreast in all matters of this nature.

Despite these minor weaknesses, this is a reference book of great value, one which is worthy of consultation by both young and old practitioners, when information on pathology is required.

—F. E. Helmore.

THE ARTICULATOR, 1952, Journal of Sydney University Dental Undergraduates' Association, Vol. 9, No. 10, Sydney.

The 1952 edition of this Journal, produced by the Sydney University Dental Undergraduates' Association, more than upholds the high standard of previous issues. Sometimes students sacrifice literary values with over emphasis upon sophistication and risque humour without art. Editor Hiatt is to be congratulated upon an annual presentation of student journalism of superior quality.

For those who may doubt the outlook of the present day student *The Articulator* is commended. There is nothing wrong with the embryo dentist's scale of values. They are realistic. In the journal there is scant respect for the pomps and vanities, the small affectations of life. This is a healthy sign in the young.

Speaking of the dentist, the editor has this to say "With the background of a sound scientific training, he is in a position to render a valuable service to the community irrespective of whether he is called a "Technician," "Professional man" or "Scientist." This is well put. In the manner of living many are wrongly classified. It is an attitude of mind which really counts.

A much to be encouraged feature for undergraduate attention is the A.D.A. Prize for the best contribution to *The Articulator*. Donovan's "A Cert for Saturday" is a particularly good story. Style, atmosphere and progression of theme show a literary craftsmanship of unusual promise. Dental literature will be the better for those trained in journalism.

One feels younger for the reading of *The Articulator*. With all its buoyancy, hope and good values there is much of quality coming into dentistry. A reviewer therefore does not altogether accept the title of a chapter devoted to individuals of the Final Year. It has been called "And So We Say Farewell." The journal itself and the personalities recorded are an assurance that good things shall survive; for them there can be no "farewell."

—John H. Wilson.

INTRAVENOUS ANAESTHESIA IN DENTISTRY, by S. L. Drummond-Jackson, London, 1952, Staples Press. (152 pp.). Price 47s. 0d. *Our copy by courtesy of the publishers.*

This book is a monograph based on a thesis delivered by the author to the F.D.I. Congress of 1952. Couched in lecture note style it is devoid of illustrations of any sort. It describes the author's experience with intravenous anaesthesia as a sole anaesthetic agent for multiple cavity preparation. Subsequently the patient is allowed to recover consciousness and the restorations proceeded with. Only a pharyngeal sponge is used as a barrier to the ingress of foreign bodies to the air passages and lungs. Thus the onus is on the operator not to drop anything down the pharynx, and the absolute protection against the entry of foreign material that a full endotracheal technique confers is ignored.

The author, while tending to discredit nasal nitrous oxide and oxygen, omits mention of the fact that nitrous oxide, because of its inherent weakness, is the only agent that can be relatively safely used without an endotracheal technique, since it permits continuous preservation of the cough reflex and there is

with it no depression of respiration or respiratory centre. In contrast all intravenous anaesthetic agents depress respiration and simultaneously by their parasympathetico mimetic action hypersensitise tthe larynx to spasm. The airway obstruction caused by either the laryngeal spasm or a foreign body is thus added to an already depressed respiration—a dangerous combination. The risks of the anoxia produced by this combination of circumstances acting on a respiratory centre already depressed by the intravenous agent is insufficiently stressed by the author. He lightly dismisses laryngeal spasm as almost nonexistent and advocates for its correction the old Biblical method of mouth to mouth insufflation. Useful as this is in an emergency it is surely a poor substitute for inflation of a patient's lungs with oxygen from a reservoir bag attached to a well-fitting face mask.

The book obviously condones the principles of the dentist acting as his own anaesthetist. The wisdom of this is open to grave doubt as, quite apart from the impossibility of doing two things efficiently at the same time, the possible legal repercussions in the case of misadventure places the dentist in a very indefensible position. The use of premedication is alluded to but no specific direction as to the type or amount is given. Atropine is not mentioned. The author's use of ethyl chloride to desensitise the skin prior to the prick of the thiopentone needle seems a good idea.

The technique advocated obviously gives good results in the author's hands. This is a tribute, not to the inherent safety of the technique, but to the author's undoubted skill in avoiding its pitfalls. The unskilful imitator who gaily drops amalgam past a pharyngeal packing will soon find himself with an immediate laryngeal spasm or a delayed pulmonary abscess. The young graduate with a flimsy training in general anaesthesia should be particularly cautioned against thinking that the method offers him a safe and sure anaesthetic procedure, attractive as it may seem at first sight. He must realise that the method is a potentially dangerous one and only possible of good results and relative safety with the very special organisation and skill of the author. It is regretted therefore that the book cannot be recommended as illustrative of a safe anaesthetic technique suitable for general routine use.

—A. P. Balthasar.

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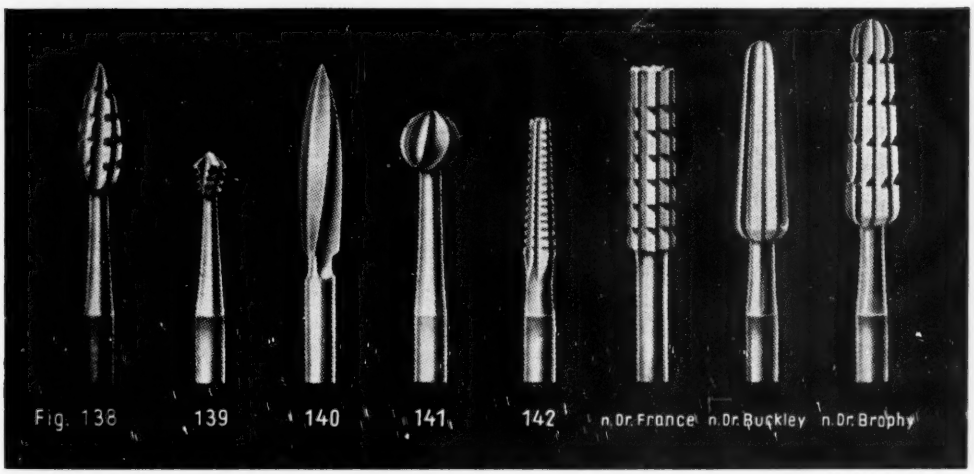

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